



Sports and Exercise Medicine, Centre for Experimental Medicine and
Rheumatology, William Harvey Research Institute, Queen Mary
University of London.

Biomechanical factors associated with previous hamstring injury in high level sprinting athletes

Submitted in part fulfillment of the requirements of the degree of Doctor of
Philosophy

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i. Statement of Originality

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Collaborators:

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ii. Abstract

Hamstring injury is common in sprinting sports and injury recurrence remains a major concern. The aim of this thesis is to explore the biomechanical characteristics of athletes following sprint related hamstring injury.

We conducted 1) An examination of already published research on biomechanical deficits following hamstring injury in athletes who had returned to sport by means of a systematic review and meta-analysis; 2) A detailed examination of sprinting following hamstring injury in athletes who had returned to sport muscle activity using 3D motion capture and surface EMG; 3) An examination of high intensity eccentric loading performance in previously injured athletes using low density, high surface area surface EMG and measures of force; 4) An analysis of hamstring muscle recovery until return to sport following hamstring injury using low density, high surface area surface EMG and measures of force via case reports in two elite athletes.

Previous research indicates that athletes who had returned to sport following hamstring injury continue to display deficits in force production, especially during slow eccentric contractions. The observational studies indicate that athletes run with significantly asymmetric movements about the pelvis and hip that would place their hamstrings under increased length during the terminal swing phase of sprinting. Furthermore, significant alterations in late swing EMG ratios suggest relatively reduced activity in the previously injured biceps femoris. Spatial activation of the hamstring appears altered in previously injured athletes, with reduced relative activation of the proximal muscle and reduced median frequency values in the medial muscle compared to control limbs. Asymmetries in activation patterns are also noted in the pre-return to sport phase.

Previous hamstring injury is associated with significant alterations in force production, movement symmetry and muscle activation patterns following return to sport highlighting the complexity of this injury and the need for advanced rehabilitation screening approaches.

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viii. Abbreviations

ACPSEM	Association of Chartered Physiotherapists in Sports and Exercise Medicine
AFL	Australian Football League
APT	Angle of Peak Torque
BF/BiFem	Biceps femoris muscle
C/contra	Contralateral
CC	Case control
CH	Cohort
CI	Confidence interval
CS	Cross sectional
Deg/s; °/s	Degrees per second
EAI	Epidemiological Appraisal Instrument
EMG	Electromyography/Electromyographic
EO/ExtObl	External oblique muscle of the abdomen
ES/ErecSpin	Erector Spinae Muscle
FA	Football Association
FIFA	Fédération Internationale de Football Association
GAA	Gaelic Athletic Association
GM/GlutMax	Gluteus Maximus muscle
H:Q	Hamstring to quadriceps ratio
HQ, MQ, LQ	High, medium, low quality
I/ipsi	Ipsilateral
Iemg	Integrated EMG
LH	Long head
MDF	Median frequency
Mdn	Median
NP	Not present
PATS	Postural agility and trunk stability
PD	Pooled data
POLICE	Protect, Optimal Loading, Ice, Compression , Elevation
PRICE	Protect, Rest, Ice, Compression, Elevation
QMUL	Queen Mary University of London
REFA	EMG data collection hardware
RICE	Rest, Ice, Compression, Elevation
RTP	Return to play
SMD	Standard mean difference
STSM	Semitendinosus/semimembranosus
Subj	Subject
UCD	University College Dublin
UEFA	The Union of European Football Associations
UI	Uninjured included

1 Introduction

1.1 Background

Hamstring injuries are common in sports which involve high speed running. Re-injury rates are high even after rehabilitation and successful return to sport. A more thorough understanding of the biomechanical characteristics of athletes with a history of sprint related hamstring injury may provide insight into post injury dysfunction and highlight key areas to be addressed during rehabilitation. The extent of the problem of sprint related hamstring injury will be presented with reference to available epidemiological research. Data related to injury mechanisms and consequences will be provided and best practice with regard to injury prevention and rehabilitation will be outlined. Through this process, current gaps in understanding and scientific knowledge in relation to hamstring injury will be illustrated. Finally, the content of this thesis will be outlined with specific reference to the value of this research in enhancing the understanding of post-injury hamstring dysfunction.

1.2 Epidemiology of hamstring injury

Repeated epidemiological studies highlight the high frequency of sprint related hamstring injury across single and multiple seasons in range of sports including multiple football codes, track and field sports, cricket and hurling (1-10). Indeed, hamstring injury is one of the most common injuries across all of these sports, accounting for between 6 and 37% of all injuries recorded (11, 12). In terms of population prevalence, hamstring injuries occur in 21-22.4% of individuals in a given season across a range of sports (8, 10, 12-14). The incidence of hamstring injury ranges from 1.3 per 1000 match playing hours in elite European football (14) to 5.6 per 1000 match playing hours in English rugby union (12). Athletes lose between 16 and 26 days from sport as a result of this injury (12, 13, 15). Of particular note is that the incidence of hamstring injury appears to be increasing in recent years (13, 14), despite apparent improvement in diagnosis and rehabilitation.

1.3 Hamstring injury risk factors

Risk factors for hamstring injury are multifactorial and include non-modifiable factors such as increasing age and ethnicity, as well as modifiable factors such as deficits in pre-season strength, flexibility, fatigue and match/performance congestion (16-22). Of particular clinical relevance is that a prior history of hamstring injury is the leading risk factor for future hamstring re-injury and is associated with a 2 to 6 fold increase in risk (11, 13, 23)

even when other risk factors are accounted for. One study highlighted an 11.6 fold increase (95% CI 3.5-39) in re-injury risk in elite association football during the season following a hamstring injury (2). Re-injury rates have been reported to be as high as 12-63.3% across multiple sports when measured over one or two subsequent seasons (11).

It is increasingly recognised that risk factors in hamstring injury are complex and multifactorial (23). This challenges the clinician to consider multiple and often complex inter-related issues when designing injury prevention and/or rehabilitation programmes. Deficits in lumbo-pelvic-hip control and flexibility have been identified as potential risk factors in hamstring injury (23). Running with increased peak anterior pelvic tilt, hip flexion and knee extension theoretically leads to increased peak strain on the biceps femoris during terminal swing (24, 25).

Increasing age has been highlighted as risk factor in hamstring injury (11, 20). This is true even when other confounding factors such as previous injury are considered. It is not currently clear why athletes display up to a 4 fold increased risk of hamstring injury as they progress into their mid-twenties. Suggested reasons include natural age related reductions in muscle volume but it seems less likely that this would apply to elite level 24 year-old athletes. Another possible explanation is in the tentative links between hamstring muscle dysfunction and lower lumbar degenerative changes (26, 27). Perhaps tethering of the nerve root or maladaptive postural control linked to spinal dysfunction could have a bearing on hamstring function. These links however, require further investigation and remain speculative.

Silder et al (28) have reported a proliferation in scar tissue in the region of the musculotendinous junction following injury leading to significantly increased levels of strain across the whole muscle especially in adjacent muscle fibres during eccentric contractions. Should these levels of strain exceed the mechanical limits of these fibres an injury is likely to occur (29, 30). Early and effective post-injury rehabilitation may be key in minimising excessive scar formation (30, 31).

Untreated strength asymmetry in preseason professional footballers has a significant association with subsequent hamstring injury (relative risk = 4.66; 95% confidence interval: 2.01-10.8) (32). Specifically subjects presenting with at least 2 of the following asymmetries in torque around the knee were considered to be at significantly increased risk of injury: bilateral asymmetry of >15% in peak concentric hamstring torque (at 60 deg/s or 240 deg/s), bilateral asymmetry of >15% in eccentric hamstring torque (at 30 deg/s or 120

deg/s); concentric Hamstring/Quadriceps ratio of <0.47 (at 60 deg/s or 240 deg/s) and a mixed Hamstring eccentric (30 deg/s)/Quadriceps concentric (240deg/s) ratio of <0.89 . Pre-season asymmetries in knee flexor strength as well as imbalances in hamstring/quadriceps peak torque (H:Q ratio) have also been suggested as potential predictors of subsequent hamstring injury in American and association football, AFL and track athletics (17, 19, 22, 32-35). However other papers have found no association between asymmetry and injury risk (19, 20). These conflicting findings illustrate the complexity in examining for strength imbalances/deficits in hamstring injury prevention.

Hamstring injuries tend to occur late in each half of European football games and have recently been associated with games congestion within UEFA competitions (2, 36). Both factors suggest that fatigue and injury are associated. This may be related to inherent physiological consequences within the muscles or more systemic effects. Fatigue has been shown to lead to increases in knee extension and decreases in hip flexion during running (37). In addition, fatigue appears to lead to a selective loss of eccentric hamstring torque generation capability (38-42).

The persistently high re-injury rates seen in hamstring injury suggest that current rehabilitation approaches may be inadequate. However, we currently lack a clear understanding of what characteristics persist in the previously injured athlete which are resistant to rehabilitation. Current diagnostic approaches are heavily based on radiological imaging, particularly MRI. However, MRI findings are poorly correlated with re-injury rates (43) and athletes often re-injure in the absence of any radiological evidence of persistent structural abnormality. Through exploring more detailed characteristics, more detailed indicators of re-injury risk may be identifiable. This information may inform more successful rehabilitation approaches and be used to develop enhanced screening and diagnostic tests to guide rehabilitation and inform return to sport decision making.

1.4 Hamstring injury mechanisms

During high speed running, the hamstrings are almost constantly active, contracting concentrically to pull the leg posteriorly through stance and acting eccentrically to slow the forward moving distal limb at the end of swing (44, 45). Although the muscle complex as a whole is active within both the concentric and eccentric phases, the largest contribution comes from the biceps femoris (46, 47). Sprint related hamstring injury risk is thought to be predominantly confined to the biceps femoris (long head) (48). A growing body of research

points to the terminal swing phase of sprinting as the moment when the biceps femoris is likely to injure (49-51) (Figure 1).

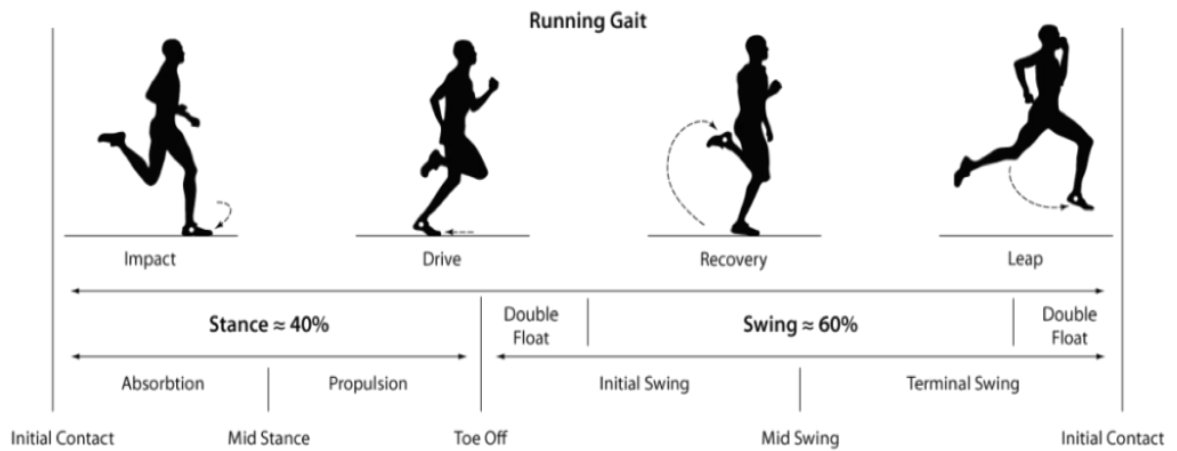


Figure 1: Phases of the running gait cycle (52).

During terminal swing the hip is flexed and the knee is nearing terminal extension. The lengthening biceps femoris is acting eccentrically to slow the tibia down in preparation for foot contact. In this range (nearing full extension), the biceps femoris moment arm is rapidly decreasing and the muscle is losing biomechanical advantage (see Figure 2) (53). For the same mechanical effect, much higher eccentric forces need to be created near end range knee extension. The risk of injury lies in the combination of a lengthened muscle generating high eccentric forces. The magnitude of force generated by the biceps femoris is proportional to the velocity of the running gait. Indeed, Chumanov et al (46) examined differences between submaximal (80%) and maximal running speeds, finding that although peak stretch of the hamstring did not change, peak net hamstring force increased from 36 N/kg to 52 N/kg (i.e. a 38% increase), and the average net negative work increased from 1.4 to 2.6 J/kg (i.e. 86% increase). The large increase in energy per unit mass (i.e. eccentric workload) between sub-maximal and maximal running underpins the mechanism of high speed hamstring injury. When this workload exceeds the maximum tolerance levels the muscle structure is acutely damaged (25, 44, 45, 49, 51).

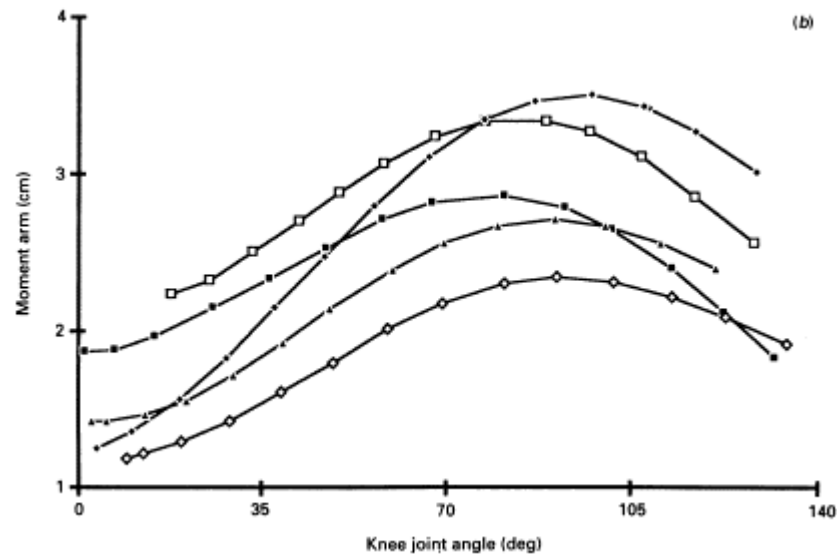


Figure 2: Moment arm of the biceps femoris through knee joint range in 5 cadavers (53).

This thesis is focussed on the biomechanical characteristics in athletes following high speed running associated injury which typically affects the biceps femoris musculotendinous junction. Askling (54) proposed an alternative slow stretching type injury which occurs at the extreme end of range of the hamstring when the hip is flexed and the knee is forced into end range extension e.g. during high kicking movement in dance. This injury typically involves the proximal semimembranosus and is associated with a more prolonged rehabilitation period (55). Although somewhat arbitrary and simplified, this dual classification system is referred to frequently within sports medicine literature and serves as a basic guide to differing presentations in clinical practice.

1.5 Hamstring injury consequences

At a physiological level, when the shear forces exceed the maximum tolerance of muscle, the damage which occurs is not only confined to the myofibre. Adjacent structures including the endomyseal, perimyseal and associated blood vessels may tear leading to the formation of a haematoma. In a more severe tear, the epimysium may be compromised and the haematoma may extend into the intermuscular and subcutaneous tissues and present as bruising over the posterior thigh (29, 30, 56). The post-injury muscle repair process is characterised by a four overlapping phases: bleeding, inflammation, proliferation, and remodelling (see Figure 3) (57). The intrinsic cellular mechanisms which drive these process are responsive (positively and negatively) to extrinsic influence. Additionally, the healing tissues are vulnerable (29, 30) and exposure to the correct magnitude of loading at the correct time is key in ensuring optimum healing and in

preventing re-rupture. A clear understanding of what, when, and how to load forms the cornerstone of current rehabilitation practice (58-60).

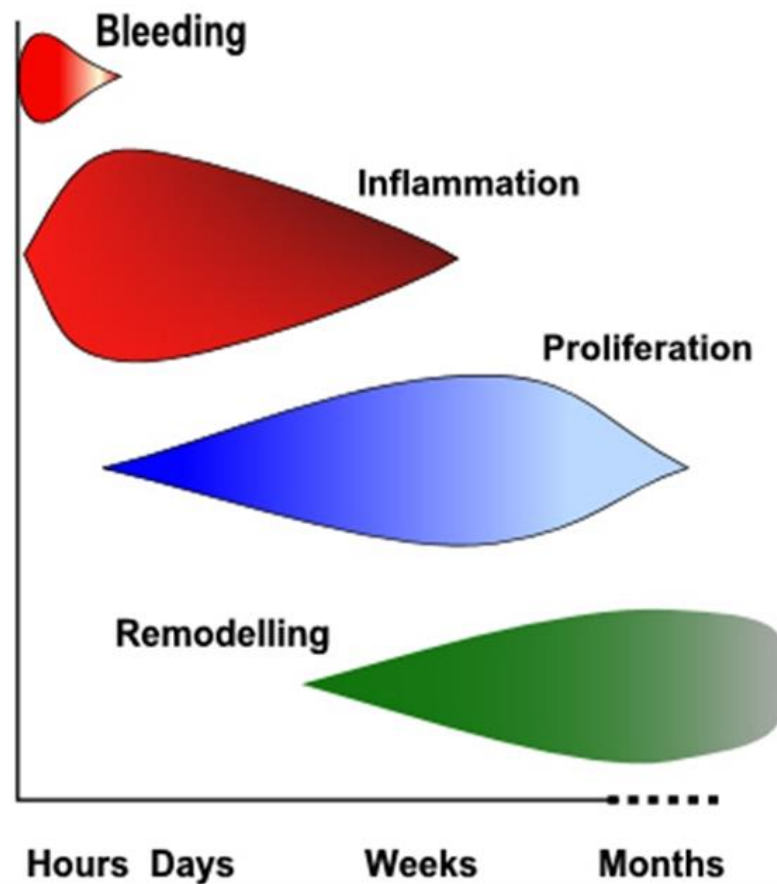


Figure 3: Graphic representation of the soft tissue healing process (57).

Magnetic resonance imaging can provide excellent detail about the location, extent and progression of the haematoma and resultant inflammatory process. In the region between to two opposing ends of torn muscle, the rapidly phagocytosed haematoma is progressively replaced with a scar. Early granulation tissue (laid down via the cross linking of circulating fibrin and fibronectin) forms a scaffold for fibroblasts. These fibroblasts begin to produce the proteoglycans and proteins of the extracellular matrix to restore the structural integrity of the muscle. The initial substances produced by the fibroblasts (e.g. collagen type III) lack tensile strength and are vulnerable to re-rupture. The strain resistant collagen type I is laid down a few days later and this process continues for several weeks. The scar becomes more injury resistant than the surrounding tissue after about 10 days (29, 30).

The regenerating myofibres pierce the end of the scar from either side and grow inwards towards the centre of the injury site. Early mechanical stimulus is needed to ensure that the lateral borders of the uninjured myofibres in the two muscle stumps attach firmly to their surrounding extracellular matrix. This firm adhesion prevents the muscle stumps from pulling apart during contraction and enables the transmission of mechanical stress across the scar site. The combination of new myofibre ingrowth and maturation of the scar site leads to a gradual reduction in the size of the scar. It is thought that the scar never completely resolves and remains as a thin collagen sheath between opposing and, eventually, interlaced muscle stumps (29, 56).

1.6 Hamstring injury rehabilitation

At the tissue level, successful hamstring injury rehabilitation requires a carefully progressed exposure to mechanical stimulus while protecting the vulnerable tissues from re-injury (58-60). A clear understanding of the process outlined in the previous section, enables the rehabilitator to design and implement the correct strategies at the right time. Suboptimal or incomplete recovery may result from both overly cautious or overly aggressive rehabilitation (61). Prolonged immobilisation following muscle injury is associated with significant muscle atrophy, increased amounts of collagen within the extracellular matrix and prolonged recovery times. Conversely overly early and/or aggressive mobilisation and loading can result in excessive scar formation, poor penetration of new myofibrils into the scar and run the risk of re-injury (29, 30, 56). It appears that the early scar tissue needs a period of relative immobilisation to develop sufficient resilience to withstand the application of force by the contracting muscle (29, 30). The duration of immobilisation will depend on the severity of the injury i.e. the magnitude of fibre disruption. Following this, a carefully progressed loading programme is required both to stimulate regeneration and restore strength and function (58, 59). Indeed, early mobilisation is associated with improved outcomes at a structural level including more parallel arrangement of regenerating myofibrils and more rapid restoration of muscle strength (29-31, 56).

The realisation that relatively early exposure to mobilisation and loading is optimal following soft tissue injury, has led to changes in practice in recent years. This is most clearly reflected in alterations to the acronyms used to promote adherence to best practice and convey a message to the wider public. The commonly advocated response to soft tissue injury for many years was to reduce the risk of re-injury and limit the inflammatory response. This approach was summed up in the acronym *RICE* (Rest, Ice, Compression,

Elevation) with no reference made to mobilisation or loading. Even the more recent *PRICE* (Protect, Rest, Ice, Compression, Elevation) recommendations promoted by the Association of Chartered Physiotherapist in Sports and Exercise Medicine (ACPSEM) in the UK up to 2010, made no reference to early and progressive loading (58). In 2010, Chris Bleakley and colleagues (58) reviewed the *PRICE* guidelines on behalf of the ACPSEM by carrying out a substantial review of the literature. They recommended, that following a short period of unloading, progressive mechanical loading should definitely be initiated after the acute phases. For the majority of soft tissue injuries, complete tissue unloading beyond the acute phases of injury should definitely be avoided. In lieu of these recommendations, Bleakley et al subsequently suggested the use of the alternative acronym *POLICE* (Protect, Optimal Loading, Ice, Compression, Elevation) with optimal loading described as an incremental and balanced exposure to early mechanical load (59).

Hamstring specific rehabilitation approaches have also developed significantly over the past decade. In addition to early and progressive loading, several papers have been published which incorporate postural control and function specific elements within their rehabilitation approaches. In 2006, Sherry and Best (62) highlighted a large reduction in re-injury rates when a small group of athletes were exposed to postural agility and trunk stability type exercises compared to strengthening and stretching work alone. Indeed increased activity of the gluteus maximus and external oblique have, at least theoretically be shown to reduce peak biceps femoris strain (24, 46). Rehabilitation programmes which address postural control have been shown to be superior to those that don't in terms of injury recurrence (62, 63). Consequently, the appreciation that hamstrings appear to be influenced, and have a role in lumbopelvic and knee postural control, have led to the incorporation of postural control training in modern rehabilitation approaches (60, 62, 63). Furthermore, a more thorough understanding of the injury mechanisms involved in sprint related hamstring injury in particular, and the specific nature of adaptation to imposed demands within the human body, have resulted in the incorporation of sport specific and eccentric load focussed strategies within current rehabilitation practice (60, 63). Recent studies highlight reduced re-injury rates when recovering athletes are exposed to progressive sprint training, end range and (eventually high speed) eccentric loading combined with increasingly challenging lumbopelvic and lower limb postural control tasks (60, 62, 63). Finally, Petersen *et al* (64) highlight a large reduction in re-injury rates in athletes who were exposed to a regime of slow, high intensity eccentric training in the form of the Nordic. This data has since been confirmed with other studies (34, 64, 65). The

Nordic is a hamstring targeted body weight exercise performed by slowly lowering the chest to the ground from a high kneeling position without flexing the hips, while the ankles are held. However, the effectiveness of the Nordic poses additional questions relating to underlying its injury prevention mechanisms. The Nordic is an inner to mid-range, slow velocity contraction exercise. However, hamstring injuries occur at high speed in the outer functional range. This suggests some mechanism beyond strength gain may underlie the beneficial effects of this exercise.

1.7 Limitations in current knowledge

With a more scientific approach to injury rehabilitation, a growing understanding of the complexity of hamstring function and a better knowledge of injury mechanisms has led to significantly improved rehabilitation strategies. However, even the most recent epidemiological studies continue to provide evidence that injury and re-injury rates remain persistently high with sprint related hamstring injury (7, 23, 36, 66, 67). This suggests that there are still some questions to be answered and perhaps improvements to be made in how one approaches and monitors hamstring injury rehabilitation.

A sports science research group led by Dr. Anthony Shield and Dr David Opar in Brisbane, have recently theorised that some of the remaining answers to hamstring injury recurrence may lie in the neuromuscular aspects of post-injury hamstring function (20, 61). There is a small, but growing body of research from this group, which suggests that previously injured hamstrings may exhibit persistent inhibition of activation following return to sport using EMG and functional MRI T2 relaxation times as outcome measures (65, 68-71). This group have postulated that such post-injury inhibition may result from a combination of pain driven muscle atrophy and weakness, alongside suboptimal early rehabilitation resulting in shorter biceps femoris long head fascicle length and weakness in the outer functional range of the muscle. This outer range (eccentric) weakness leaves the biceps femoris long head vulnerable to re-injury at terminal swing during sprinting (20, 61).

It is also perhaps worth considering cortical aspects of hamstring muscle function. The biceps femoris long head is injured during eccentric loading (51, 72). The neural control of an eccentric contraction requires greater cortical involvement, not least in inhibiting activity in the antagonist muscle (73-75). There is some evidence to suggest that maladaptive cortical remapping can occur with musculoskeletal pain in the elbow extensors (76) and lumbar spine (77-79). It is possible that spinal and supra-spinal mal-adaptations may also occur following hamstring injury. Although this theory remains to be tested,

some of the mechanisms underlying neuromuscular inhibition may be also found more centrally as well as in the periphery.

Finally, current approaches to injury classification are strongly biased towards structural manifestation of injury assessed via radiological imaging (80, 81). The Munich consensus proposed an expanded classification system specific to the hamstring Table 1 by including both functional and structural subtypes. Unfortunately, this system was based on an expert discussion and lacks validity. Indeed, an early prospective study has highlighted the prognostic value of the Munich structural sub-classification in predicting recovery times but indicated that the functional sub-classification has less value in this regard (26).

Type A: INDIRECT MUSCLE INJURY/DISORDER		
FUNCTIONAL	Overexertion related muscle disorder	A. Fatigue induced muscle disorder
		B Delayed onset muscle soreness (DOMS)
	Neuromuscular related muscle disorder	A. Spine induced
		B. Muscle induced
STRUCTURAL	Partial muscle tear	A Minor
		B Moderate
	(Sub) total tear (including subtotal or complete muscle tear or tendinous avulsion)	
TYPE B: DIRECT MUSCLE INJURY		
CONTUSION		
LACERATION		

Table 1: Munich consensus for muscle injury classification (27)

Although it is increasingly recognised that hamstring injury and its consequences manifest beyond the peripheral aspects of structural muscle fibre disruption, there is a lack of tools available for screening and diagnosing specific hamstring related dysfunction following an injury. Such tools may offer clearer insight into the consequences of hamstring injury and may provide guidance in improving athlete outcomes.

1.8 Potential impact of this thesis

Successful rehabilitation of hamstring injury is characterised by two factors. Firstly, the athlete should be returned to their maximum sporting function and secondly any risk of re-injury should be minimised in so far as possible. In sprint related hamstring injury, it appears that we are less successful at achieving the latter. This suggests that there may be characteristics associated with previous injury which remain unaddressed. In the following thesis, biomechanical characteristics related to hamstring function will be explored in previously injured athletes. It is hoped that this exploration will highlight novel aspects of post-injury hamstring function, provide a target for the development of enhanced rehabilitation approaches and provide a basis to future research aimed at reducing hamstring re-injury risk.

2 Aims and hypotheses

2.1 Aims and objectives

2.1.1 Primary aim

The overarching aim of this thesis is to explore the biomechanical characteristics of athletes who have previously sustained a sprint related hamstring injury. The impact of this research will be to provide a better understanding of hamstring related function following injury and thus provide primary data for further study which may enhance current rehabilitation strategies and help reduce re-injury rates.

2.1.2 Secondary aims and objectives

1. To summarise the current evidence related to persistent biomechanical deficits following hamstring injury following return to sport. This will aid the identification of gaps within the current literature and guide the direction of the observation studies within the thesis.

Objective: To complete a systematic review and meta-analysis of current literature. Data pertaining to hamstring muscle function in previously injured athletes will be collated and compared to non-injured control data. Specifically, data related to hamstring related force production, movement control and EMG will be assessed – see Chapter 3.

2. To examine the biomechanical features of sprinting in athletes who had returned to sport following non-contact, sprint related hamstring injury in order to inform rehabilitation and prevention strategies.

Objective: To recruit two groups of athletes; one with prior hamstring injury and their closely matched uninjured teammates, and make inter-group comparisons of EMG activity levels in the biceps femoris with respect to functionally associated muscle groups, and lower limb kinematic symmetry during sprinting – see Chapter 5

3. To investigate the potential application of low density multichannel EMG in detecting post-injury neuromuscular inhibition in the hamstrings during a maximal effort eccentric loading activity (the 'Nordic').

Objective: To recruit a group of athletes a history of prior hamstring injury alongside an uninjured, closely matched control group. To make comparison of EMG activity during a 'Nordic' loading exercise using low

density arrays of EMG sensors placed over the posterior thigh.

Simultaneous measures of force will be collected – see Chapter 6

4. To explore the acute and sub-acute stages of muscle rehabilitation in elite athletes using a selection of tools including low density EMG arrays, isokinetic dynamometry and other external measures of force.

Objective: To recruit individual cases from the elite sporting population and complete a number of case reports to illustrate the recovery process following acute sprint related hamstring injury with particular regard to spatial EMG - see Chapter 7

2.2 Hypotheses

2.2.1 *Study one: Systematic Review (Chapter 3)*

The hypothesis was that there would be an association between hamstring related force, EMG and/or movement outcomes when previously injured athletes who have returned to sport are compared to uninjured controls. Specifically, it was anticipated that prior literature would indicate that previously injured athletes would present with persistent deficits in knee flexor strength, alterations in EMG activity and/or movement patterns indicative of unresolved muscle dysfunction.

2.2.2 *Study two: Observational case control (Running) (Chapter 5)*

The hypothesis was that there would be a difference between previously injured and uninjured groups with respect to muscle activation ratios and/or kinematic variables across the stride. Specifically it was anticipated that athletes with previous injured would run with increased anterior pelvic tilt and EMG patterns indicative of impaired biceps femoris activation on the previously injured limb.

2.2.3 *Study three: Observational case control (Nordic) (Chapter 6)*

The hypothesis was that there would be a difference between previously injured and uninjured groups with respect to the magnitude of EMG activity at the time of peak force and/or peak amplitude activity during a Nordic loading exercise. Specifically, it was anticipated that athletes would show evidence of impaired activation of the biceps femoris on the previously injured limb with associated compensatory activity elsewhere.

2.2.4 Study four: Case reports (Chapter 7)

The hypothesis was that there would be demonstrable asymmetries in hamstring activation patterns between the injured and uninjured limbs of participant indicative of impaired recruitments of the previously injured muscle and compensatory activation elsewhere. Measures of force would also show asymmetry, specifically reduced knee flexor strength in the previously injured limb.

3 Systematic Review

Persistent biomechanical deficits following return to sport after hamstring injury. A systematic review and meta-analysis

3.1 Introduction

Hamstring injuries are one of the most common injuries in sprint related sport. They are the most frequent injury seen in sprint athletics, association football, Gaelic football, hurling and Australian rules football (3, 4, 67, 82). Hamstring injuries are ranked second only to thigh haematomas in rugby union in terms of frequency (5). Of particular concern is that re-injury remains a major issue with prior injury consistently identified as the primary risk factor for injury recurrence (11, 20, 23, 83).

Slow eccentric loading exercises (e.g. the Nordic drop) (64), progressive exposure to sprint related exercises (63) and lumbopelvic postural control work (60, 62) have become central to current rehabilitation approaches following sprint related hamstring injury. Indeed, some encouraging data has emerged in recent years indicating better outcomes in re-injury prevention when such approaches are implemented (62-64). In recent years the theory of neuromuscular inhibition has emerged as a possible basis to explaining high recurrence rates associated with prior hamstring injury (20, 61). This theory proposes that both a combination of pain induced inhibition leading to muscle atrophy, alongside poor rehabilitation may lead to alteration in the biomechanical properties of the biceps femoris.

Evidence indicates that muscles mechanical properties are altered following hamstring injury secondary to scar volumes and muscle belly atrophy as confirmed by magnetic resonance imaging (28, 84). Indeed, suboptimal and overly aggressive rehabilitation practices ('too much, too early') may contribute to increase scar volumes as well as pain and resultant disuse atrophy. However, even in conditions of optimal rehabilitation there is further evidence of impaired activation of hamstring muscle post injury. This is provided by Bourne et al. (68) who employed functional MRI immediately following intensive eccentric loading of the hamstring muscles. They identified chronically impaired activation in previously injured muscles compared to healthy controls characterised by reduced relaxation times in the previously injured muscle.

The most obvious example of chronic functional deficits following hamstring injury is provided through measures of strength – several studies, all included in this review, have explored strength outcomes, mostly using isokinetic dynamometry, with many illustrating

persistent deficits following injury. In addition the optimal muscle length for force generation shifts to a shorter muscle lengths (16, 21). Sprint related hamstring injuries are thought to occur at terminal swing during sprinting, a phase of the running gait cycle where the biceps femoris is both lengthened and under high eccentric load as it decelerates the lower leg in preparation for ground contact (44, 50, 85-87). The shifting of the angle of peak torque to shorter muscle lengths illustrates a mechanism by which the biceps femoris is less functional during this maximal muscle length phase and therefore more susceptible to injury.

This aim of this review is to collate and present the evidence in which measures of biomechanical outcomes are made in individuals with previous hamstring injury compared to un-injured comparators. This analysis will be limited to outcomes related to strength, movement and EMG in a population who have returned to full participation in sporting activity. The value of this review may be to highlight key targets for rehabilitation and identify important screening applications which may provide clearer guidance when making return to play decisions.

3.2 Methods

3.2.1 Search strategy

MEDLINE, CINHL, Web of Science Current Contents and SportDiscus databases were searched from inception to January 2016. No date limits were placed on the search but only articles published in English were included. The search terms (Table 2) were devised in order to identify papers which met the inclusion/exclusion criteria. The reference lists of included articles were screened for any omitted studies. Furthermore, citation tracking was performed on key articles via Google Scholar to ensure maximal retrieval of suitable papers.

Biomechanic* OR electromyograp* OR EMG OR myoelectric* OR sEMG OR fine wire OR RMS OR frequency OR twitch OR %MVC OR muscle activit* OR excita* OR force OR torque OR RFD OR APT OR isokinetic dynamomet* OR isometric OR eccentric OR concentric OR strength OR power OR kinetic* OR kinematic* OR movement OR control OR symmetr* OR coordinat* OR motion OR 3D OR dimension* OR flexion OR extension OR rotation OR tilt OR valgus OR varus OR adduction OR abduction.
AND
deficit* OR change OR alteration OR loss OR gain OR increase* OR effect* OR manifest*
AND
hamstring* OR thigh OR semitendin* OR semimembran* OR "femoral biceps" OR "biceps femoris"
AND
injur* OR tear* OR rupt* OR strain*

Table 2: Search terms

3.2.2 Inclusion/Exclusion criteria

Studies which measured biomechanical characteristics in individuals with previous hamstring injury were collated. Data was limited to studies where measures were taken following return to sport/normal activity. Biomechanical characteristics were defined as kinematic, kinetic and/or EMG outcomes. For the purpose of this analysis both prospective cohort and retrospective case as well as case-control data was analysed. Any studies which failed to report prospective or comparative differences post-injury were excluded. In addition, studies which employed subjective measures of strength only were excluded.

3.2.3 Data for extraction

All data relating to force, EMG or movement outcomes were extracted. Where relevant data was unavailable for synthesis or full paper copies were unavailable, direct contact was made with the corresponding author and this data was sought. In addition, for studies which appeared to be subsets of other published datasets from the same research team, clarification of uniqueness was sought from corresponding authors to avoid duplication of

data. If the paper was confirmed to be a subset of another published dataset, the subset paper was omitted.

3.2.4 Quality assessment and risk of bias

The Epidemiological Appraisal Instrument (EAI) (88) was used to assess the quality of included studies. The EAI is a 42 item checklist which has undergone extensive validation (88). In accordance with the methodology of (89) and (90) the EAI was adjusted and items pertaining to randomisation, exposure validity/reliability and adverse events were omitted as they did not fit with the objectives of this review. This left 36 items remaining for scoring. Studies were scored for each item with full adherence scoring 2, partial adherence scoring 1 and non-adherence or where a lack of sufficient clarity led to an inability to determine adherence scoring 0. A total score was summated for each paper and an average calculated with non-applicable items omitted from the calculation. The highest average score possible was therefore 2. Studies were divided into high quality (HQ), medium quality (MQ) and low quality (LQ) using the average score as the determining factor. A HQ rating equated to a quality score of ≥ 1.4 , moderate quality 1.1 to < 1.4 and low quality (< 1.1) as per previously published methods (88, 91).

3.2.5 Synthesis of results

Extracted data was collated in table form. This included means and standard deviations pertaining to relevant data, alongside patient demographics and other data as relevant. Where data was suited to meta-analysis means, standard deviations and subject numbers were entered into a Review Manager database (Version 5.3, Copenhagen: The Nordic Cochrane Centre, The Cochrane Collaboration database). Standard mean differences (SMDs) were calculated and classified as small (≤ 0.59), medium (0.60–1.19) or large (≥ 1.20) as described previously (92) (89). Statistical heterogeneity was calculated within the Review Manager software using χ^2 and I^2 statistics which give a p value where $p < 0.05$ confirms significant heterogeneity.

Levels of evidence were defined using the criteria outlined previously (91, 93), namely:

Strong evidence: Evidence provided by pooled results derived from three or more studies, including a minimum of two high-quality studies, which were statistically homogenous ($p > 0.05$); may be associated with a statistically significant or non-significant pooled result

Moderate evidence: Statistically significant pooled results derived from multiple studies that were statistically heterogeneous ($p < 0.05$), including at least one high-quality study; or from multiple moderate- or low- quality studies, which were statistically homogenous ($p > 0.05$)

Limited evidence: Results from one high-quality study or multiple moderate- or low-quality studies that are statistically heterogeneous ($p < 0.05$).

Very limited evidence: Results from one moderate, or low- quality study.

Conflicting evidence: Pooled results that are insignificant and derived from multiple statistically heterogeneous studies ($p < 0.05$) (regardless of quality).

The robustness of the meta-analysis was checked by performing a sensitivity analysis using high and moderate quality studies only. In addition, two sub-analyses were completed to ensure the trustworthiness of the findings. Firstly, an analysis was completed on all studies that outlined a clear and evidence based rehabilitation protocol, specifically one which included eccentric loading and sport specific training. In addition, a further sub-analysis was carried out on papers which tested elite athletes only. It was anticipated that such subjects may be more likely to avail of professional rehabilitation advice following a hamstring injury. They may also be exposed to more advanced strength and conditioning within their sport. Therefore, such examining both elite and those who had competed a defined rehabilitation protocol would reduce the risk of positive findings secondary to failure to rehabilitate adequately.

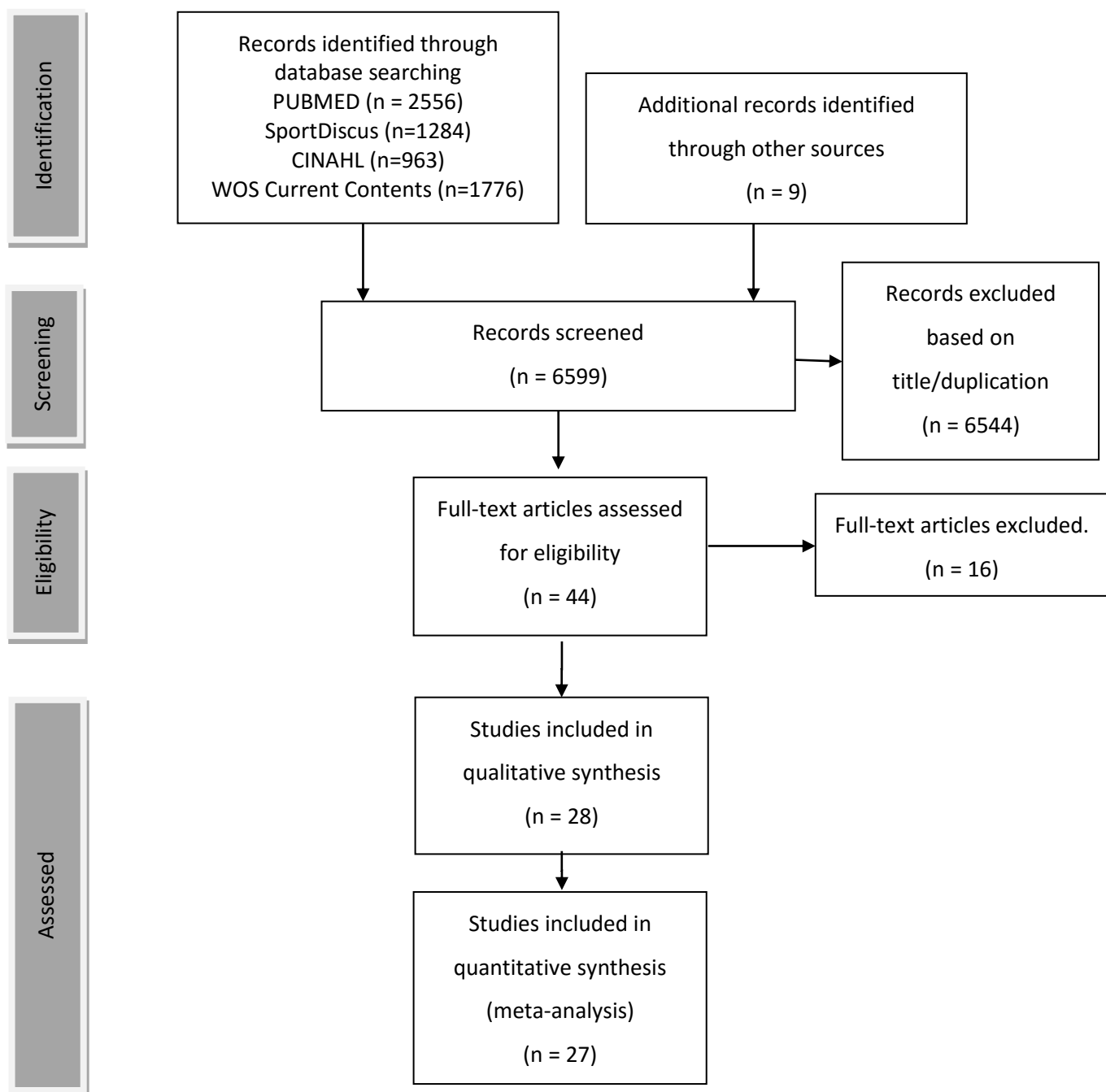


Figure 4: PRISMA flow diagram outlining study retrieval process.

3.3 Results

3.3.1 *Search outcomes*

Initially, 6599 article titles were returned from the database searches, citation tracking and reference list checking. Following assessment of title for inclusion criteria, 44 articles were further screened through abstract and/or full paper reading. A further 16 papers were omitted leaving 28 papers as meeting the stated inclusion criteria (see Figure 4). The primary reason for omitting articles was the lack of post-injury testing using those outcomes outlined in the inclusion criteria (28, 62-64, 94-98). Other reasons included a lack of comparison between injured and uninjured conditions (32, 34, 68, 99) studies where outcomes were related to interventions only (100) and studies where injured subjects had not returned to sport (54).

3.3.2 *Characteristics of included articles.*

3.3.2.1 *Study design*

Of the 28 studies included for synthesis, 13 employed a case control design (69-71, 101-110) with close matching of injured and uninjured participants for secondary confounders. A further 10 studies employed a cross sectional design (16, 111-118) with 5 including uninjured (and unmatched) subjects within their analysis (16, 111, 115, 117, 118). Finally, 5 articles are presented as cohort studies (43, 65, 84, 119, 120), with one including data from uninjured participants in their analysis (65). Demographic information for included studies is presented in Table 3.

3.3.2.2 *Subject/injury characteristics*

In all included articles, previously injured subjects had returned to unrestricted physical activity. Only seven studies (43, 65, 69-71, 84, 120) describe a structured rehabilitation process with four papers (69-71, 84) making reference to the progression outlined by Heiderscheit et al (60), one (43) referring specifically to a 'Postural Agility and Trunk Stability (PATs)' programme as per Sherry and Best (62) and finally, two referring to progressive loading, sport specific exercises and eccentric loading as being the key principles of rehabilitation (65, 120). In terms of diagnostics, eight papers reported a confirmed diagnosis with MRI scan (43, 65, 71, 84, 111, 119-121), while two employed ultrasound (112, 113). One paper (103) reported partial use of MRI (44% of cases) while 14 papers report that a diagnosis was based on either medical and/or subjective patient report only (69, 70, 101, 104-106, 108-110, 115-119, 121). Injury and rehabilitation descriptors for each study are outlined in Table 4.

3.3.2.3 *Sporting characteristics*

Subjects were recruited from across a range of sports. These are outlined in Table 3.

3.3.2.4 *Time since injury/RTP*

All subjects had returned to participation in their sport and the time of testing. The minimum amount of time post injury was 7 days (120) while the maximum follow-up period was up to two full seasons post injury.

3.3.2.5 *Quality/risk of bias assessment.*

Following appraisal with the EAI, two studies were classified as HQ (65, 120), five as MQ (43, 71, 106, 111, 114) and the remaining 21 as LQ (Table 5). This indicates that the majority of articles meeting the inclusion criteria scored poorly on methodological quality.

Authors	Design	Subject Numbers	Age Mean (SD/Range)	Height Mean (SD)	Weight Mean (SD)	Gender (M:F)	Sport
Barreira et al 2015	CC	6 pre-injured; 11 controls	24.5 (2.3); 21.3 (1.2)	1.79(0.03); 1.83(0.03)	76.3(2.5); 82.2(2.8)	17:0	Elite association football
Brughelli et al 2010	CC	11 pre-injured; 11 controls	22.4 (NR); 21.9 (NR)	183.0 (NR); 183.5 (NR)	84.1 (NR); 84.0 (NR)	22:0	Semi-pro Australian rules football
Brockett et al 2004	CS + UI	9 pre-injured; 18 controls	NR (22-38); NR (19-28)	NR	NR	16:1	Elite/sub elite Australian rules football and track
Cameron et al 2004	CS + UI	7 pre-injured; 13 controls	23.6 (3.2) PD	185.5 (8.5) PD	87.8 (9.1) PD	20:0	Elite Australian rules football
Croisier et al 2004	CS	26 pre-injured	25 (8)	1.8 (0.07)	74 (7)	26:0	Elite association football, track, martial arts
Croisier et al 2000	CS	23 pre-injured	24 (7)	1.78 (0.08)	72 (8)	23:0	Elite association football, track, martial arts
Daly et al 2015	CC	9 pre-injured; 8 controls	28.3 (5.2); 25.0 (5.3)	1.79 (0.06); 1.82 (0.05)	81.2 (6.6); 84.3 (7.0)	17:0	Elite hurling
Dauty et al 2004	CC	11 pre-injured; 17 controls	23.2 (3.1); 23.0 (3.8)	180 (7.6); 176.7 (5.5)	76.2 (8.0); 72.5 (7.0)	28:0	Elite association football
De Vos et al 2014	CH	64 pre-injured; 16 no f/up	28 (22-32); 28 (23-33)	NR	NR	61:3 15:1	Various
Doherty et al 2012	CS	42 pre-injured (16 elite)	20.64 (1.51)	175.93 (10.94)	81.77 (18.33)	28:14	Various university sports
Jonhagen et al 1994	CS+UI	11 pre-injured; 9 controls	22 (18-29); 22.2 (19-26)	NR	NR	20:0	Sprinting – sub 11.32s 100m
Lee et al 2009	CS	12 kinematics; 14 isokinetics	23.6 (2.8)	184.4 (4.9)	86.6 (10.6)	14:0	Various
Mackey et al 2011	CC	9 pre-injured; 9 control	23.89 (4.57); 20.44(1.13)	1.76 (0.05); 1.79 (0.04)	81.17 (8.14); 80.11 (11.47)	18:0	Club level Gaelic Football
Mendiguchia et al 2014	CC	14 pre-injured; 14 controls	21.9 (2.5); 21.6 (2.5)	174.6 (4.7); 173.5 (4.7)	69.3 (5.9); 72.4 (7.1)	28:0	Semi-pro association football
O'Sullivan et al 2008	CS+UI	15 pre-injured; 29 controls	21.2 (1.8) PD	180.5 (6.6) PD	82.1 (8.6) PD	44:0	University Gaelic football
O'Sullivan and Burns 2009	CS+UI	8 pre-injured; 13 controls	20.7 (3.3); 20.3 (2.69)	NR	60.7 (5.7); 63.9 (9.9)	0:20	University Gaelic football
Opar et al 2013 (a)	CS	20 pre-injured	NR	NR	NR	20:0	Elite Australian rules football, rugby union, track
Opar et al 2013 (b)	CC	13 pre-injured; 15 controls	26.2 (5.8); 26.7 (5.8)	1.8 (0.04); 1.8 (0.05)	83.0 (14.8); 83.5 (7.9)	28:0	Recreationally active
Opar et al 2013 (c)	CC	13 pre-injured; 13 controls	26.6 (5.8) ; 25.9 (3.4)	1.8 (0.04); 1.8 (0.05)	83.2 (14.3); 82.8 (7.5)	26:0	Recreationally active
Opar et al 2015 (a)	CH+UI	17 pre-injured; 82 controls	23.3 (2.6); 22.6 (3.3)	186.2 (6.5); 188.3 (7.6)	85.9 (6.6); 87.8 (7.6)	99:0	Elite Australian rules football
Paton et al 1989	CC	7 pre-injured; 7 controls	19.9 (17-27).	NR	NR	14:0	Elite association football
Sanfilippo et al 2013	CH	25 pre-injured	24 (9)	170 (0.5)	73.8 (25.8)	20:5	Recreationally active
Silder et al 2010	CH	18 pre-injured	24.3 (9.98)	NR	NR	14:4	50% track, others various
Sole et al 2011	CC	15 pre-injured; 15 controls	24.6 (5.1); 22.5 (4.9)	1.77 (0.06); 1.82 (0.05)	81.5 (13.0); 80.0 (12.1)	30:0	Various
Sole et al 2012	CC	16 pre-injured; 18 controls	24.8 (5.2); 22.6 (5.0)	176.9 (6.5); 183.0 (5.1)	79.1 (8.4); 80.7 (12.0)	34:0	Various
Timmins et al 2015	CC	16 pre-injured; 20 controls	23.7 (3.3) ; 26.1 (7.4)	1.85 (0.07); 1.80 (0.05)	83.6 (7.9); 78.1 (8.7)	36:0	Injured = various elite; Controls = recreational
Tol et al 2014	CH	52 pre-injured	24.9 (18-38)	NR	NR	52:0	Elite association football
Worrell et al 1991	CC	16 pre-injured; 16 controls	20.7 (NR) PD	182.74 (NR) PD	82.23 (NR) PD	32:0	Various

Table 3 Characteristics of included articles: study design, subject demographics and sport (CC: case control, CS: cross sectional study, CS + UI: cross sectional study with uninjured subjects included, CH: cohort study, CH + UI cohort study with uninjured subjects included. Age, height and weight are expressed as means and standard deviation/ranges where available and are subdivided for previously injured and controls as appropriate. NR = not reported; PD= reported as pooled data across groups).

Authors	Muscle injured	Grade	Diagnostic confirmation	Time Absent	Mechanism	Rehabilitation	Timing of testing v injury/RTP
Barreira et al 2015	Not specified	G1 (n=3), G2 (n=3)	Medical report	20.3 (2.2) days	Sprinting	Not specified (one club)	141- 518 days
Brughelli et al 2010	Not specified	G1-3	Not specified	>7 days	Not specified	Not specified (different clubs)	Post return to sport.
Brockett et al 2004	Not specified	G1-3	Not reported	>1 week training	2 kick; 7 sprint	None with eccentric.	1 month to previous season
Cameron et al 2004	Not specified	Not specified	MRI and medical report	>1 match	Not specified	Not specified - one club	>12 weeks
Croisier et al 2004	'Typically' BF	Not specified	U/S	Not specified	Not specified	Not specified	Range: 3-12 months
Croisier et al 2000	Not specified	Not specified	Clinical and U/S	Not specified	Not specified	Not specified	Range: 2-12 months
Daly et al 2015	Not specified	Not specified	44% MRI; 88.8% medical report	>48 hrs	Sprinting	Not specified (different clubs)	11.17 (9.2) months
Dauty et al 2004	Not specified	8 moderate (7-30d) 7 severe (30+days)	Medical report	7+ days	Not specified	Not specified (different clubs)	10.8 (6.5) months
De Vos et al 2014	BFLH 56 STSM 8 BFLH 13 STSM 3	G1=18 G2=46 G1=5, G2=11	MRI	40 (31–55) days 46 (33–67) days	Sprinting: n=47	Well described progression (PATS)	Initial assessment @ 3 (2–4) days. Post RTP @ 3 (2–5) days
Doherty et al 2012	Not specified	G1-2	Not specified	Not specified	Not specified	'Some rehab'	<24 months but back to sport
Jonhagen et al 1994	Not reported	Moderate–Major	Subj report	~ 2 months	Sprinting	Not reported	Within previous 2 seasons
Lee et al 2009	Not specified	G2.3 (0.4)	Medical report	Not specified	Not specified	Not specified	19 (12.5) months
Mackey et al 2011	Not specified	Not specified	Subj report	>7 days	Not specified	Not specified	>6 weeks < 1 year
Mendiguchia et al 2014	Not specified	Not specified	Subj report	3.5 (1.5) weeks	Non-contact	Not specified	<4 weeks post RTP
O'Sullivan et al 2008	Not reported	Not reported	Subj report	3.5 (1.8) weeks	Not reported	Not specified	>12 weeks
O'Sullivan & Burns 2009	Not reported	Not reported	Subj report	>7 days	Not reported	Returned to full participation	>6 weeks – 2 years
Opar et al 2013 (a)	BFLH (75%)	G1–3; G1=65%	Medical report & MRI	Not reported	Sprinting 80%	Not specified	Mdn= 3.9 (1.0-18.2 months)
Opar et al 2013 (b)	BF (100%)	Not specified	Medical report	3.6 (1.2)	Not specified	Largely as per Heiderscheit	<12 months (Mean = 5.7m)
Opar et al 2013 (c)	BF (100%)	G2	Medical report	Median 4 weeks	Not specified	Largely as per Heiderscheit	5.3(4.3) months
Opar et al 2015 (a)	BFLH (76.5%)	Not reported	MRI	38.6 (21.7) days	Running (76.5%)	Emphasis on eccentrics & high speed running	22.9(12.3) days
Paton et al 1989	Not reported	>G2	Not reported	Not reported	Not reported	Not reported	< 1 year
Sanfilippo et al 2013	BF (66.7%)	Not reported	MRI	26 (17-49)	Not specified	As per Heiderscheit	@ RTP and 6 months @ 6 months
Silder et al 2010	BF (100%)	Not reported	Subj/medical report MRI	Not reported	Not reported	2 weeks of rehab min & RTP	7.2(1.99) months
Sole et al 2011	Not reported	Not reported	Subj and medical report	3.9 (1.9) weeks	Not specified	Not specified	3.6 (3.5) months
Sole et al 2012	Not reported	Not reported	Subj and medical report	4.3 (2.1) weeks	Not specified	Not specified	3.7 (3.5) months
Timmins et al 2015	BFLH	Not reported	MRI	Not reported	Not specified	As per Heiderscheit and eccentrics	< 18 months but back to sport
Tol et al 2014	Not reported	52% G1 48% G2	MRI	21 days (7–43)	Not specified	Standardised protocol: progressive load, Sport specific elements and eccentrics	21 days (range 7–43) – following 6 stage process
Worrell et al 1991	Not reported	Not reported	Subjective report	15.2 (9.3)	Not specified	Not specified	<18 months but back to sport

Table 4: Injury characteristics, rehabilitation completed and timing of testing in relation to injury data for included studies (BF: biceps femoris, LH: long head, STSM: semitendinosus/semimembranosus, G: Grade, Subj: subjective, RTP: Return to play, Mdn: Median, PATS: Postural agility and trunk stability)

	Hypothesis, aims and obj.	Exposure	Outcome	Study design	Source	Eligibility	Participation	Characteristics	Lost to f/up	Individual	Environmental	Stats	Clear description	Random variability	Stats parameters	Sample size calc	Group comparability	Participation rate	Time period	Subject losses	Incident cases	Repeatability	Baseline considered	Observer blinding	Reliability	Validity	Comparability	Observation period	Individual	Environmental	Sufficient follow-up	Adjustment for follow-up	Outcome x exposure	Subgrouping for outcome	Sufficient participation rates	Sufficient random sampling	Overall Score (max 62)	Average score (max 2)	Quality (HQ/MQ/LQ)
Tol et al 2014	Y	Y	Y	Y	P	Y	NA	P	Y	P	N	Y	P	Y	N	N	NA	NA	Y	Y	NA	Y	N	UTD	Y	Y	NA	NA	N	N	Y	N	N	N	UTD	UTD	32	1.6	HQ
Opar et al 2015 (a)	Y	P	Y	Y	Y	Y	Y	Y	Y	P	N	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	N	UTD	Y	UTD	Y	Y	N	N	Y	N	N	N	Y	Y	52	1.44	HQ	
Cameron et al 2004	Y	P	Y	P	Y	Y	P	Y	Y	P	N	Y	Y	Y	Y	N	Y	Y	Y	Y	Y	Y	Y	UTD	UTD	Y	Y	Y	P	N	Y	N	N	N	Y	UTD	49	1.36	MQ
Doherty et al 2012	Y	P	Y	Y	Y	Y	NA	Y	Y	P	Y	Y	Y	Y	N	N	NA	UTD	UTD	Y	NA	P	NA	UTD	Y	Y	Y	NA	P	Y	Y	N	N	N	N	UTD	38	1.19	MQ
De Vos et al 2014	Y	Y	Y	Y	P	Y	NA	Y	Y	P	N	Y	Y	Y	Y	N	NA	UTD	UTD	Y	Y	Y	N	UTD	UTD	UTD	NA	Y	P	N	Y	Y	N	N	UTD	UTD	39	1.18	MQ
Timmins et al 2015	Y	P	Y	Y	P	Y	NA	Y	Y	P	N	Y	Y	Y	Y	Y	UTD	NA	UTD	Y	N	Y	N	UTD	Y	Y	Y	UTD	N	N	Y	N	N	N	UTD	Y	40	1.18	MQ
Mendiguchia et al 2014	Y	P	Y	Y	P	Y	N	Y	Y	Y	Y	Y	Y	Y	Y	N	Y	UTD	P	Y	N	Y	N	UTD	Y	Y	Y	UTD	P	N	Y	N	N	N	UTD	UTD	40	1.11	MQ
Sanfilippo et al 2013	Y	Y	Y	Y	P	Y	NA	Y	Y	P	N	Y	Y	Y	N	N	NA	NA	UTD	Y	N	Y	N	UTD	Y	Y	Y	NA	P	N	Y	N	N	N	UTD	UTD	35	1.09	LQ
Lee et al 2009	Y	P	Y	Y	P	Y	NA	Y	Y	Y	N	Y	Y	Y	N	Y	NA	NA	UTD	Y	N	P	N	UTD	UTD	Y	Y	UTD	N	N	Y	N	N	N	UTD	Y	33	1.09	LQ
Barreira et al 2015	Y	P	Y	Y	Y	Y	P	Y	Y	P	N	Y	Y	Y	N	N	Y	UTD	Y	Y	N	Y	N	UTD	UTD	P	Y	Y	P	N	Y	N	N	N	UTD	UTD	39	1.08	LQ
Dauty et al 2004	Y	P	Y	Y	Y	Y	N	Y	Y	P	N	Y	Y	Y	Y	N	Y	UTD	UTD	Y	N	Y	N	UTD	UTD	Y	Y	Y	P	N	Y	N	N	N	UTD	UTD	39	1.08	LQ
Croisier et al 2002	Y	Y	Y	Y	Y	Y	NA	Y	Y	P	N	Y	P	Y	N	N	NA	NA	UTD	Y	NA	Y	NA	NA	UTD	Y	NA	NA	N	N	Y	N	N	N	UTD	UTD	30	1.07	LQ
Silder et al 2010	Y	Y	Y	Y	P	Y	NA	Y	Y	P	N	Y	Y	Y	N	N	NA	NA	UTD	Y	N	Y	N	UTD	Y	Y	Y	NA	P	N	Y	N	N	N	UTD	UTD	34	1.06	LQ
Sole et al 2011	Y	P	Y	Y	P	Y	N	Y	Y	P	N	Y	Y	Y	Y	N	Y	NA	UTD	Y	N	Y	N	UTD	Y	Y	Y	UTD	N	N	Y	N	N	N	UTD	UTD	37	1.06	LQ
Daly et al 2015	Y	P	Y	Y	Y	Y	N	Y	Y	Y	N	Y	Y	Y	N	N	Y	UTD	UTD	Y	N	Y	N	UTD	UTD	Y	Y	Y	P	N	Y	N	N	N	UTD	UTD	38	1.06	LQ
Mackey et al 2011	Y	P	Y	Y	Y	Y	P	Y	Y	Y	N	Y	Y	Y	N	N	Y	UTD	UTD	Y	N	Y	N	UTD	Y	Y	Y	UTD	N	N	Y	N	N	N	UTD	UTD	38	1.06	LQ
O'Sullivan et al 2008	Y	P	Y	Y	Y	Y	Y	Y	Y	P	P	Y	Y	Y	N	N	Y	UTD	UTD	Y	N	P	N	UTD	Y	Y	Y	UTD	N	N	Y	N	N	N	UTD	UTD	38	1.06	LQ
O'Sullivan & Burns 2009	Y	P	Y	Y	Y	Y	Y	Y	Y	P	N	Y	Y	Y	N	N	Y	UTD	UTD	Y	N	P	N	UTD	Y	Y	Y	UTD	N	N	Y	N	N	N	UTD	UTD	37	1.03	LQ
Croisier et al 2000	Y	P	Y	Y	Y	Y	NA	Y	Y	P	N	P	P	P	N	N	NA	NA	UTD	Y	NA	Y	NA	NA	UTD	Y	NA	NA	N	N	Y	N	N	N	UTD	UTD	28	1	LQ
Brughelli et al 2010	Y	P	Y	Y	P	Y	P	Y	Y	P	N	Y	Y	Y	N	N	Y	UTD	UTD	Y	N	Y	N	UTD	UTD	UTD	Y	Y	N	N	Y	N	N	N	UTD	UTD	34	0.94	LQ
Opar et al 2013 (b)	Y	P	Y	Y	P	Y	NA	Y	Y	P	N	Y	Y	Y	Y	N	P	UTD	UTD	Y	N	P	N	UTD	UTD	Y	Y	UTD	N	N	Y	N	N	N	UTD	UTD	33	0.94	LQ
Sole et al 2012	Y	P	Y	Y	N	Y	N	Y	Y	P	N	Y	Y	Y	Y	N	UTD	NA	UTD	Y	N	P	N	UTD	Y	Y	Y	UTD	N	N	Y	N	N	N	UTD	UTD	33	0.94	LQ
Opar et al 2013 (a)	Y	P	Y	Y	P	Y	NA	N	Y	N	N	Y	Y	Y	Y	N	NA	NA	UTD	Y	NA	Y	N	UTD	Y	UTD	NA	UTD	N	N	Y	N	N	N	UTD	UTD	28	0.9	LQ
Brockett et al 2004	Y	P	Y	Y	P	Y	N	Y	Y	N	N	Y	Y	Y	N	N	N	UTD	UTD	Y	N	Y	N	UTD	UTD	Y	Y	Y	N	N	Y	N	N	N	UTD	UTD	32	0.89	LQ
Opar et al 2013 (c)	Y	P	Y	Y	P	Y	NA	Y	Y	P	N	Y	Y	Y	N	N	P	UTD	UTD	Y	N	P	N	UTD	UTD	Y	Y	UTD	N	N	Y	N	N	N	UTD	UTD	31	0.89	LQ
Worrell et al 1991	Y	P	Y	Y	P	Y	NA	N	Y	N	N	Y	P	Y	N	N	P	NA	UTD	Y	N	N	N	UTD	UTD	UTD	Y	UTD	N	N	Y	N	N	N	UTD	UTD	24	0.71	LQ
Paton et al 1989	Y	P	Y	Y	P	P	N	N	Y	N	N	Y	P	N	N	N	Y	UTD	UTD	Y	N	N	N	UTD	Y	Y	Y	UTD	N	N	UTD	N	N	N	UTD	UTD	24	0.67	LQ
Jonhagen et al 1994	Y	N	Y	Y	N	P	NA	N	Y	N	N	Y	Y	Y	P	N	NA	UTD	UTD	Y	N	N	N	UTD	UTD	Y	Y	UTD	N	N	UTD	N	N	N	UTD	UTD	22	0.65	LQ

Table 5: Epidemiological Appraisal Instrument (EAI) findings ordered by quality (HQ in green; MQ in yellow and LQ in red)

3.3.3 *Kinetic Outcomes*

3.3.3.1 *Isokinetic Outcomes*

Results from several angular velocities were available and data were grouped according to speed. In two studies (117, 118) both absolute (Nm) and relative (Nm/kg) outcome were reported. Only the latter is included in the meta-analysis to avoid duplication. Sanfillipo et al (84) made comparisons between subjects at return to sport (RTS) and at 6 months following RTS. Only the 6-month data is included in the meta-analysis with the RTS data reported separately (see 3.3.3.1.7). Mackey et al (105) made comparisons within an injured population (limb x limb) and as well as between injured and uninjured groups. Only the intra-group comparisons are included within the meta-analysis with inter-group comparisons reported separately (see 3.3.3.1.7).

3.3.3.1.1 Peak torque (concentric knee flexors)

Eleven reportable studies (16, 69, 84, 105, 108, 110, 111, 115-118) compared concentric knee flexor peak torque between previously injured and uninjured conditions. Croisier and Crielaard (112) also report concentric knee flexor peak torque but provide graphical data only. Means and standard deviations were requested but were not available. There is very limited evidence (based on one study (115)) indicating a moderate reduction in peak torque values at a 30°/second contraction velocity (SMD -1.12 95% CI -2.08, 0.16). There was no significant effect of prior injury on peak torque values at any other contraction velocity (see Figure 5).

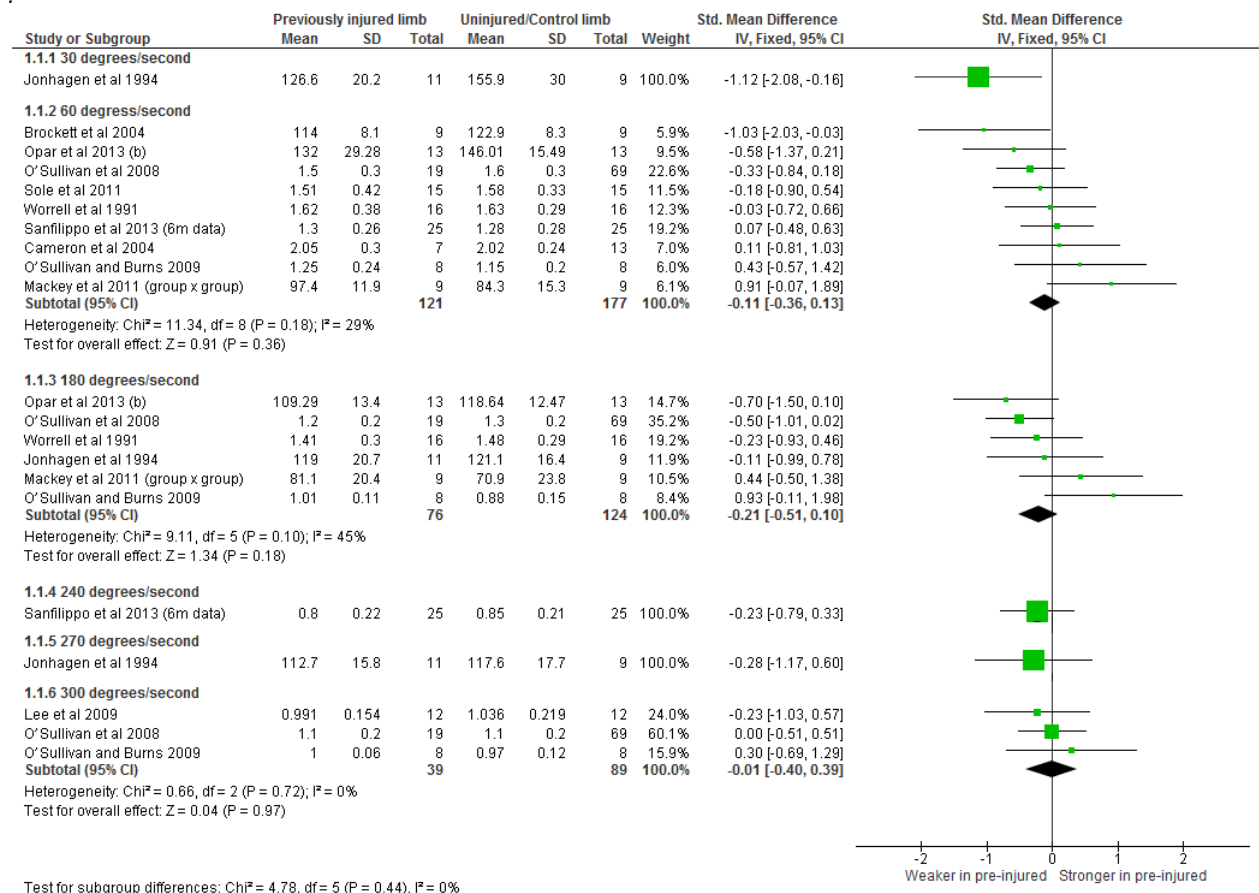


Figure 5: Forest plot showing the association between previous hamstring injury on peak concentric knee flexor torque

3.3.3.1.2 Peak torque (eccentric knee flexors)

Eight studies (69, 105, 108, 110, 114-116) explored peak torque values between previous injured and uninjured athletes during an eccentric knee flexor effort. Again, mean and standard deviation values were not available for Crosier and Crielaard (112) so this data is omitted from the meta-analysis. There is moderate evidence indicating medium reductions in eccentric peak torque at a 30°/second ($p=0.03$, SMD -0.78 95% CI -1.18, -0.07) velocity. In addition, a single study (115) provides very limited evidence of large reductions (SMD -1.84 95% CI -2.93, -0.76) in eccentric peak torque at a 230°/second contraction velocity. There are no significant findings at any other velocities (Figure 6).

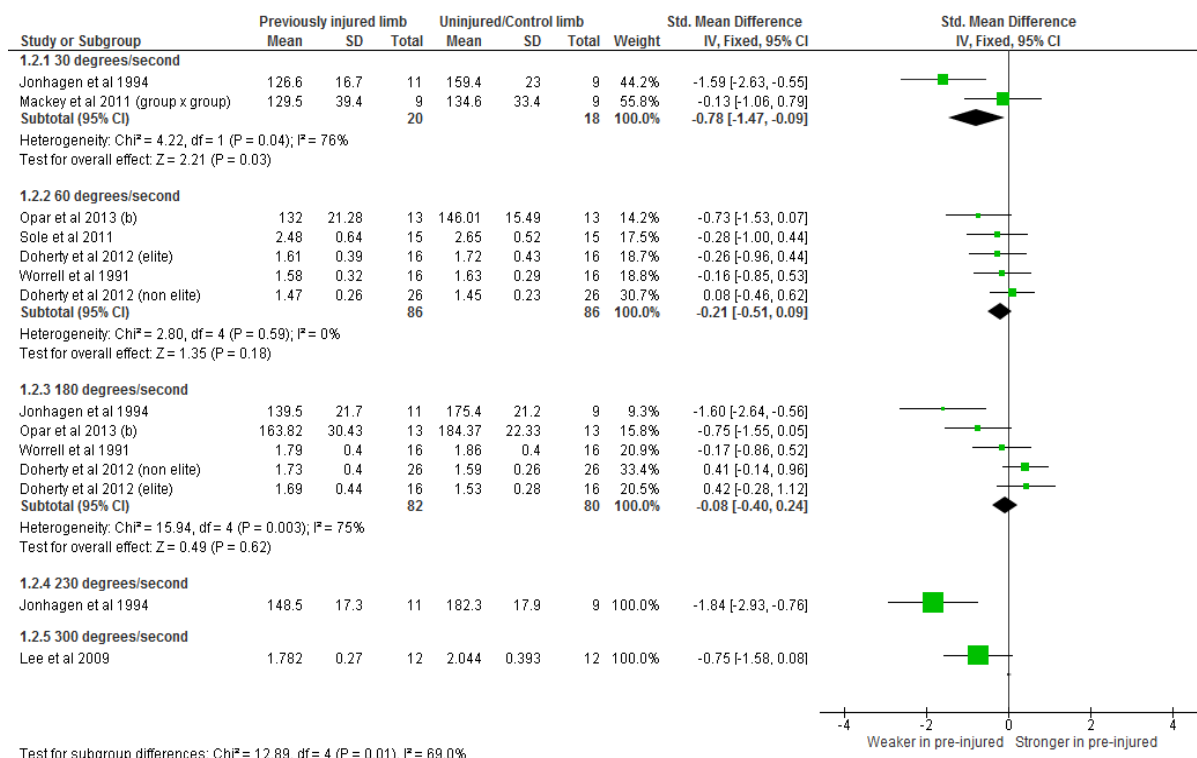


Figure 6: Forest plot outlining the effect of a previous hamstring injury on eccentric knee flexor peak torque.

3.3.3.1.3 Peak torque (concentric knee extensors)

Meta-analysis indicated no association between peak concentric knee extensor torque and prior injury to the hamstring. One study (115) provides very limited evidence for a medium reduction in torque at a 30°/second contraction velocity (SMD -1.16, 95% CI -2.13, -0.19) (Figure 7).

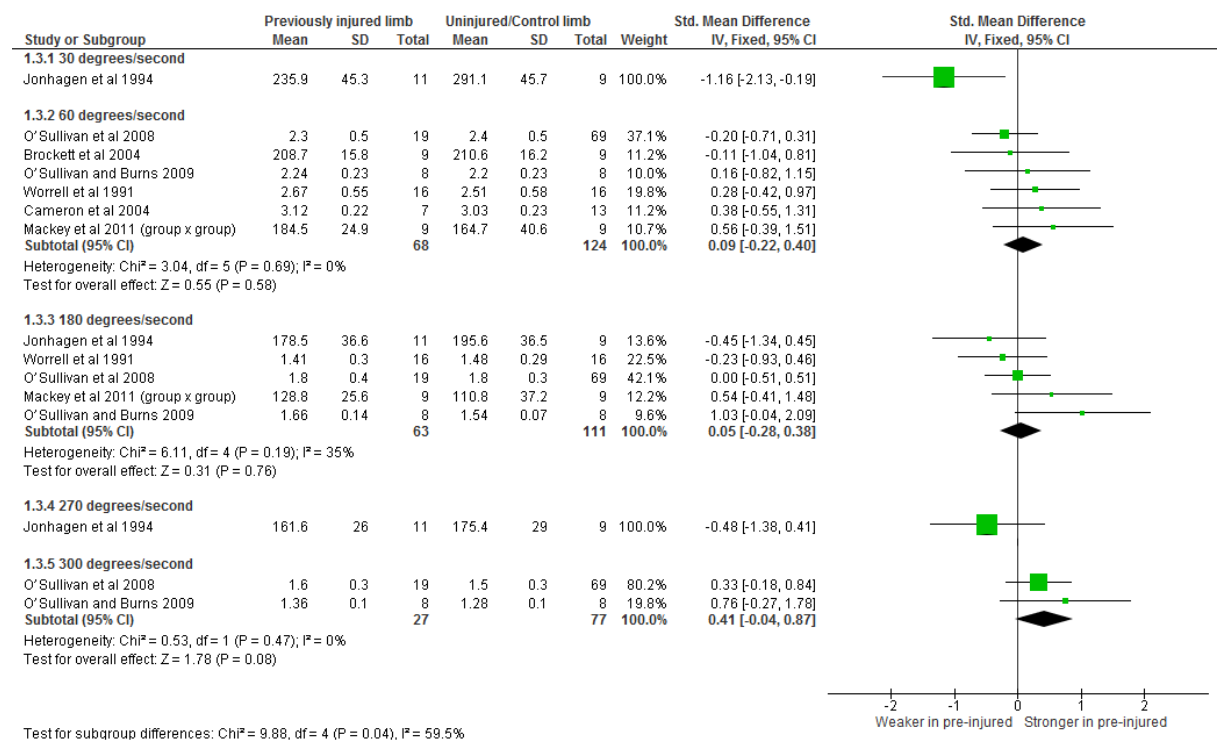


Figure 7: Forest plot outlining measures of peak concentric knee extensor torque between injured and uninjured limbs.

3.3.3.1.4 Angle of peak torque

There is no evidence of alterations in the angle of peak torque during concentric knee flexor contractions in previously injured athletes (Figure 8). This finding is based on two studies (16, 84) Mackey et al (105) provide very limited evidence of a moderate increase in angle of peak torque (towards shorter hamstring muscle lengths) when measured during an eccentric contraction (SMD 0.77, 95% CI -0.20, 1.74) (Figure 9).

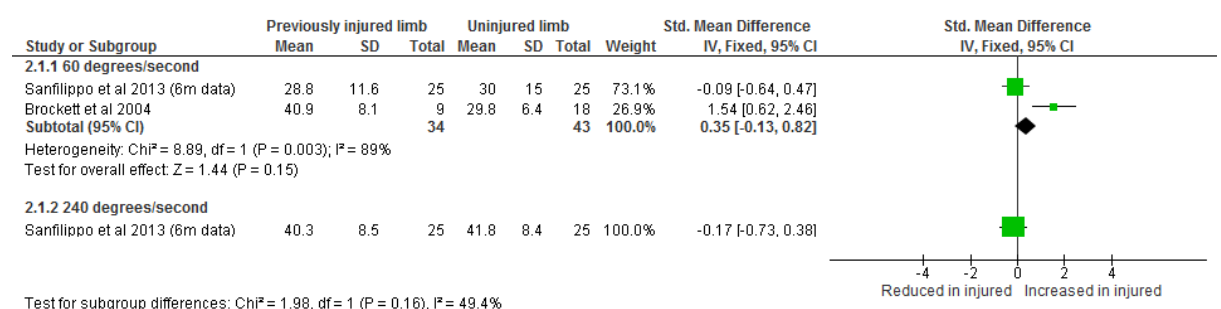


Figure 8: Angle of peak torque (concentric knee flexors).

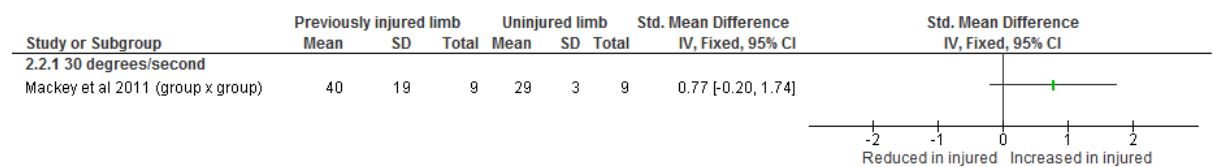


Figure 9: Angle of peak torque (eccentric knee flexors).

3.3.3.1.5 Ratio of concentric knee flexors to concentric knee extensors

Eleven studies (104, 105, 107, 108, 110, 111, 114, 117, 118) explored the ratio of concentric knee flexor to concentric knee extensor peak torque between previously injured and uninjured conditions. Mean and standard deviation values were not available for Croisier and Crielaard (112) and Croisier et al (113) so this data is omitted. There is moderate evidence indicating a small reduction in ratio values at a 60°/second:60°/second contraction velocity ($p=0.002$, SMD -0.34 95% CI -0.56, -0.12). There are no other significant findings at any other contraction velocity.

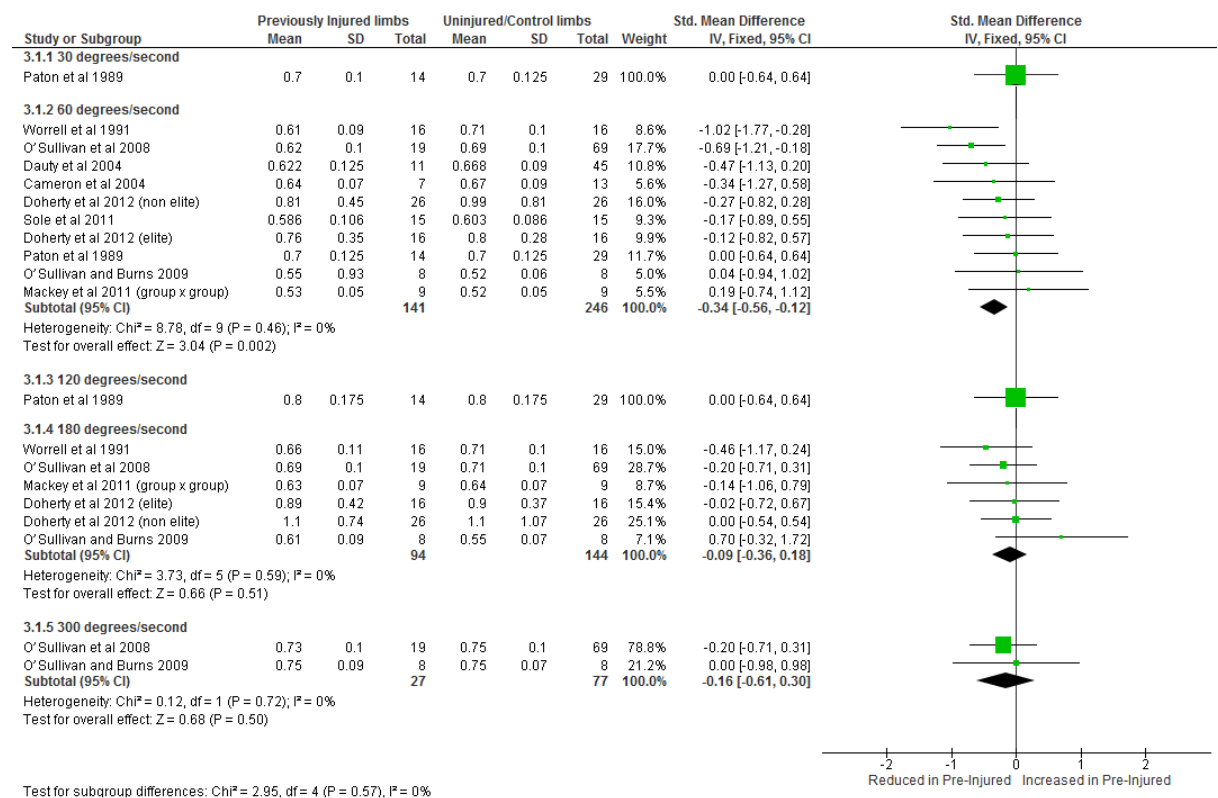


Figure 10: Concentric knee flexor to concentric knee extensor ratio.

3.3.3.1.6 Ratio of eccentric knee flexors to concentric knee extensors

Seven studies (84, 104, 105, 108, 112-114) explored the ratio of eccentric knee flexor to concentric knee extensor peak torque (i.e. the functional ratio) between previously injured

and uninjured conditions. There is moderate evidence of a medium reduction in this ratio in previously injured limbs ($p=0.0002$), SMD -0.62 95% CI -0.95, -0.29) (Figure 11) during a 60°/second:240°/second contraction velocity. A borderline significant ($p=0.05$) small reduction is noted at a 60°/second:60°/second velocity (SMD -0.29, 95% CI 0.60, 0.01).

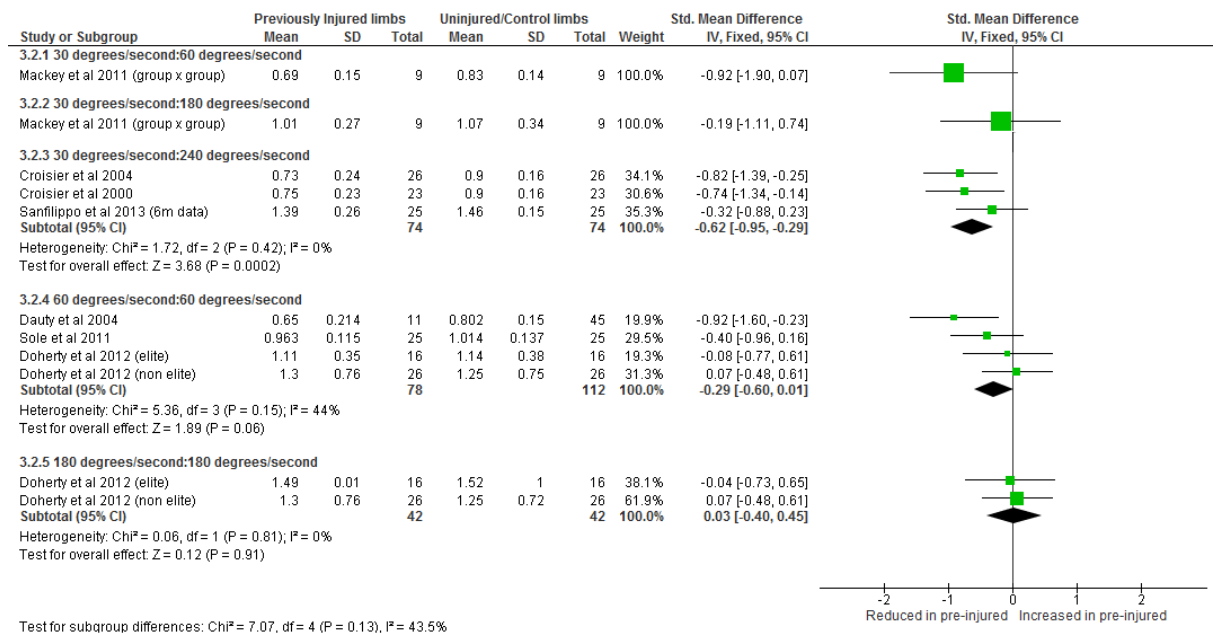


Figure 11: Eccentric knee flexor to concentric knee extensor ratio

3.3.3.1.7 Isokinetic outcomes excluded to avoid data duplication

In addition to the sixth month post RTS outcomes described above, Sanfilippo et al (84) made measurements in athletes at the time of RTS. They provide very limited evidence that subjects with previous hamstring injury had a medium reduction in functional ratio values (eccentric hamstring peak torque at 30°/second versus concentric quadriceps peak torque at 240°/second; SMD - 1.10 95% CI -1.70, -0.50). This reduction had improved by 6 month follow-up testing (see Figure 11). Mackey et al (105) provide very limited evidence of a medium size increase in angle of peak knee flexor torque (towards shorter muscle lengths) when previously injured limbs are compared to contralateral limbs in previously injured individuals (SMD 0.98 95% CI -0.01, 1.97). All other outcome tested for both these papers provide very limited evidence of small to negligible changes in SMD between test groups.

3.3.3.1.8 Other Isokinetic Outcomes

There was a large variability in methods used to explore direct and derived measures of force across included articles. This included measures of peak torque asymmetry (69, 112, 113, 119), average peak torque normalised to relative peak torque presented across time quartiles (108), calculations of force impulse, work done and rate of torque development (70) and various ratios of peak torque within and across limbs for both concentric and eccentric contractions (16, 104, 110, 115, 117-119). In order to present data in a concise and understandable way, this data was omitted from the meta-analysis.

Tol et al (120) provide evidence of highest methodological quality in this review. They explored isokinetic outcomes but limited their analysis to a description of the frequency of asymmetries >10% between the previously injured and uninjured limbs in 52 elite footballers who had completed a rehabilitation programme. They confirmed a >10% asymmetry in peak torque in 39% of participants during a concentric knee flexor contraction at 60°/s, 29% of participants during a concentric knee flexor contraction at 300°/s and 28% of participants during an eccentric knee flexor contraction at 60°/s. These authors also found that re-injury at 2 months was not associated with isokinetic deficits.

3.3.3.2 Other force related measures

3.3.3.2.1 Kinetics

Four studies explored kinetic outcomes during running (101, 102, 106, 116). Lee et al (116) used inverse dynamic calculations to examine 3 dimensional joint moments during swing phase. Barreira et al (101) calculated vertical and horizontal forces via a partially instrumented treadmill. Brughelli et al (102) calculated vertical ground reaction forces via an instrumented treadmill alongside horizontal forces at the subjects' centre of mass via an instrumented belt. Finally, Mendiguchia et al (106) extrapolated kinetic outcomes using a combination of radar telemetry, environmental and anthropometric data. Given the wide range of methodology employed, pooling of data is not possible and SMD and 95% confidence interval values are reported for individual studies only.

Of the two studies that explored external instrumented force measures during treadmill running only. Brughelli et al (102) found a large inter-limb (versus non-injured limb)(SMD - 3.81 95% CI -5.3, -2.31) and inter-group (versus dominant SMD -2.23 95% CI -3.34, -1.12; versus non-dominant SMD -1.77 95% CI -2.79, -0.75) difference in horizontal force

production at the centre of mass on the previously injured limb. This evidence is very limited as it is based on one LQ study. Conversely Barreira et al (101) provide very limited evidence of no significant inter-limb or inter-group difference in propulsive or steady state horizontal forces when measured at treadmill level. Lee et al (116) provide very limited evidence of no significant asymmetries in sagittal peak moments or powers at the hip or knee during swing in previously injured subjects. They did however highlight a small increase in sagittal knee moment during late swing which approached significance although the effect is minimum (SMD 0.26 95% CI -0.56, 1.05). Finally, Mendiguchia et al (106) (MQ) carried out two stage testing, firstly at return to sport post hamstring injury and secondly ~2months after. In their own analysis, they employed a semi-qualitative statistical method using magnitude based inferences based on effect size calculations. They found deficits in acceleration ability characterised by increased time to reach 5m, 10m and 40m during a maximal sprint. In addition, they identified deficits in power, peak horizontal force and peak velocity during sprinting in previously injured subjects. All deficits, with the exception of peak horizontal velocity had resolved by ~2 months post return to play. This very limited evidence indicates a medium reduction in peak horizontal force production in previously injured subjects at return to play (SMD -0.81 95% CI -1.59, -0.04) and a medium reduction in maximum horizontal power in previously injured subjects at return to play (SMD -0.93 95% CI -1.72, -0.15).

3.3.3.2.2 Hand-held dynamometry

Only deVos et al (43) explored the symmetry of isometric knee flexion force generation between previously injured and uninjured limbs using hand-held dynamometry. This very limited evidence indicates asymmetries in isometric force production with knee flexed at 15° and 90° of 1(16)N and 2(15)N respectively between previously injured and uninjured limbs. SMD analysis of effect was not possible due to reporting of inter-limb difference in force only.

3.3.3.2.3 NordBord

Three studies explored force generation using a NordBord, a device specifically designed to measure eccentric force generation capacity while the subject performs an eccentric loading exercise known as the Nordic drop (65, 71, 121). When inter-limb comparisons are considered, moderate evidence indicates a borderline significant and small reduction in

force values on the previously injured limb ($p=0.05$, SMD -0.38 95% CI -0.77, 0.00) (see Figure 12).

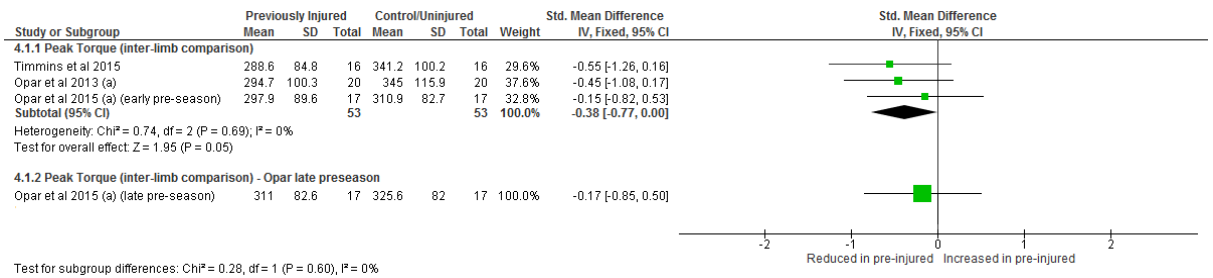


Figure 12: Previous injured limb versus uninjured limb comparisons within a previously injured population using the NordBord

When comparisons are made to control subjects, there is moderate evidence of a medium reduction in peak force between previously injured limbs and the right legs ($p=0.0002$, SMD -0.64 95% CI -0.98, -0.30) as well as a small reduction versus the left limbs ($P=0.009$, SMD -0.45 95% CI -0.79, -0.12) of control subjects (see Figure 13).

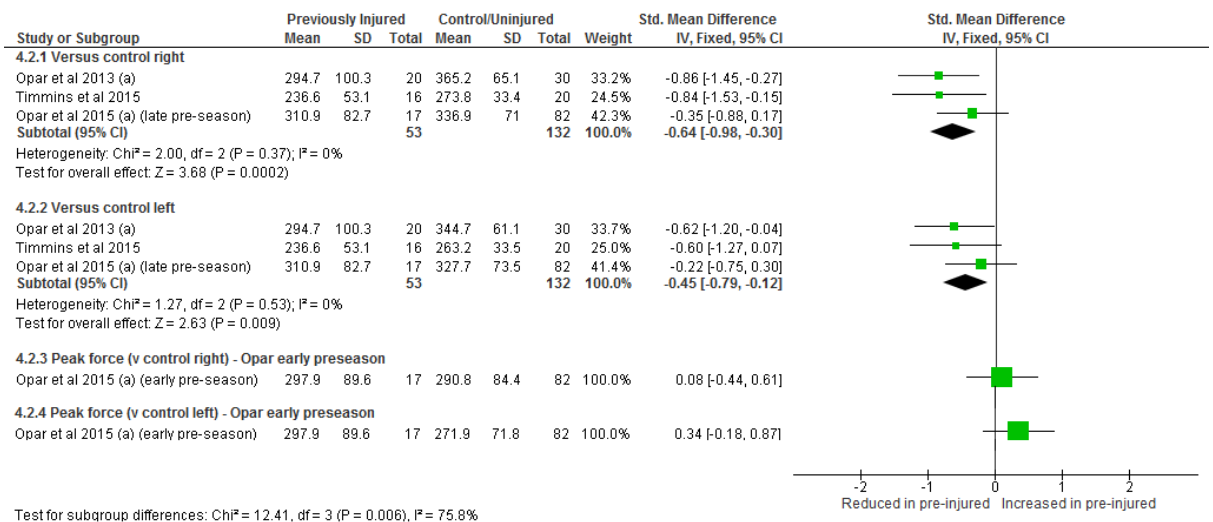


Figure 13: NordBord comparisons between previously injured limbs and control limbs (subdivided according to side)

Opar et al (65) further explored changes in strength across a preseason timeframe and provide very limited evidence that a previously injured group showed resistance to strengthening via eccentric training. This was indicated by a smaller increase in peak force (13.9(55.0)N) versus the control group (54.6(78.5)N) when assessed during the early and late pre-season using the NordBord (SMD -0.54 95% CI -1.07, -0.01).

3.3.4 Movement Outcomes

3.3.4.1 Temporal outcomes

Three studies report temporal aspects of human movement in previously injured athletes (102, 106, 109). Mendiguchia et al (106) provide very limited evidence of impaired early acceleration in previously injured athletes. This is characterised by a medium increase in time to reach 5 metres during a maximal sprint in footballers with previously hamstring injured when compared to their uninjured teammates (SMD 1.06 95% CI 0.26, 1.85). This deficit was present shortly after RTP but had resolved at follow-up testing ~2 months after. Brughelli et al (102) provide very limited evidence indicating no alteration in contact time during non-motorised treadmill running (at 80% of maximum effort) between limbs in previously injured athletes (SMD -0.1 95% CI -0.98, 0.74) and between previously injured athletes and controls (versus left: SMD -0.1 95% CI -0.93, 0.74 $P=0.82$; versus right: SMD -0.57 95% CI -1.42, 0.29 $P=0.19$). Finally, Sole et al (109) provide very limited evidence of faster initiation of movement on the previously injured limb versus control subject average, when asked to move from double leg standing to single leg standing on visual cue (-54ms (Range -108 to 0)). As these authors report differences only, SMD calculations are not possible.

3.3.4.2 Kinematic outcomes.

Four studies (102, 103, 116, 119) explored movement (i.e. kinematic) based outcomes in subjects with prior hamstring injury during high speed running. Of the four, only two report three dimensional joint movements during motorised treadmill running (103, 116). Silder et al (119) also report musculoskeletal model based hamstring muscle lengths during motorised treadmill running, while Brughelli et al (102) explored centre of mass displacement using an external rig fixed to the subjects waist during non-motorised treadmill running. Given the differing methodologies employed, pooling of data is not appropriate. All studies are considered separately and each provide very limited evidence owing to poor methodological quality.

Daly et al (103) found increased movement asymmetries in previously injured athletes. Clinically small but statistically significant relative increases in pelvic tilt (maximum 4°, $p=0.02$) and hip flexion (maximum 8°, $p=0.01$) were noted during late swing on the injured limb (compared to the uninjured limb) when comparisons were made to uninjured

controls. They also found stance phase increases in medial knee/tibia rotation in the previously injured subjects (maximum 6° , $p=0.03$). SMD calculations for these variables are not possible as standard deviations are not reported. In contrast, Lee et al (116) found a small reduction in peak hip flexion (1.9° , $p = 0.02$) during late swing when within group comparison was made between in previously injured and uninjured limbs (SMD -0.35 95% CI -1.15, 0.46). They found no difference in late swing knee extension (SMD 0.12 95% CI -0.68, 0.92). The remaining two studies reported no significant findings. Brughelli et al (102) reported no difference in centre of mass displacement between previously injured limbs versus uninjured limbs and between previously injured versus control participant limbs. Finally, Silder et al (119) report no difference in peak biceps femoris musculotendinous lengths between limbs in previously injured subjects ($p=0.36$).

3.3.5 EMG Outcomes

Six studies explored EMG activity across previously injured and uninjured conditions. Of these, two explored activity during high speed running (103, 119), three during isokinetic dynamometry (69, 70, 108) and one during a double support to single support standing manoeuvre (109). Given the different approaches used for data processing, pooling of data is not possible. SMD calculations are performed on individual studies where possible.

Differing results are obtained in the running studies. Daly et al (103) provide very limited evidence of reductions in EMG ratio values during late swing in previously injured athletes when compared to their teammates. Previously injured biceps femori appeared relatively underactive when expressed as a ratio to gluteus maximus, erector spinae and contralateral rectus femoris activity. In contrast, Silder et al (119) provide very limited evidence of no inter-limb difference in activation timings or amplitudes (normalised to mean muscle activity of the entire gait cycle and expressed as average root mean squared values for initial contact, propulsion, early swing and late swing phases) in the biceps femoris, medial hamstrings, rectus femoris and vastus lateralis muscles

Of the three studies that explored EMG outcomes during an isokinetic analysis, all examined amplitude based comparisons (69, 70, 108). All provide very limited evidence of reduced EMG activity. Specifically, one (69) reports a large reduction in normalised (to MVC) amplitude in previously strained biceps during eccentric loading (60° /second SMD -0.74, 95% CI -1.54, 0.06; 180° /second -0.66 95% CI -1.45, 0.13) when compared to the

contralateral limb in previously injured subjects. Such asymmetries were not present in uninjured subjects. Similar activation asymmetries are also described by Opar et al (70) where a small increase in asymmetry (SMD 0.38, 95% CI -0.37, 1.13) is noted in biceps femoris activity of previously injured subjects during a 180°/second eccentric contraction. Sole et al (108) confirm reduced activity for both the biceps femoris ($p=0.003$) and medial hamstring ($p=0.002$) muscles when compared to controls subjects during a 60°/second eccentric contraction especially at outer ranges of muscle length. SMD analysis is not possible for this outcome as group means and standard deviations are not presented. Opar et al (69) also explored median frequency outcomes between previously injured and non-injured subjects and provide very limited evidence of no significant differences during isokinetic testing.

Finally, Sole et al (109) provide very limited evidence that individuals with a previous history of hamstring injury had earlier onset hamstring muscle activation in preparation for a double to single leg standing manoeuvre in both the previously injured and uninjured limbs, when comparison is made to control subjects. Again, as only composite differences are reported, SMD analysis is not possible.

3.3.6 Sensitivity analysis

Only two HQ and five MQ studies are included in this review. Isokinetic peak torque outcomes are heavily based on LQ studies and there is only single paper evidence available for these outcomes. Therefore, there is very limited evidence of no change in concentric knee flexor peak torque (60°/second) in previously injured athletes following exclusion of LQ studies. Insufficient evidence of sufficient quality exists to conduct a sensitivity analysis on measures of eccentric peak torque and angle of peak torque. Moderate evidence indicates no significant changes in conventional H:Q ratio at 60°:60°/second ($p=0.23$, SMD -0.24 95% CI -0.63, 0.15). Only one MQ study reported data related to measures of functional H:Q ratio. Doherty et al (114) provide very limited evidence of no change in previously injured athletes at both a 60°:60°/second and 180°:180°/second velocity. A sensitivity analysis of NordBord outcomes indicates that there are no significant reductions in peak torque between injured and uninjured limbs of previously injured subjects (SMD -0.34 95% CI -0.83, 0.15 $P=0.17$), nor when comparison is made to uninjured controls (Right leg: SMD -0.29 95% CI -0.62, 0.03 $P=0.08$; left leg: SMD -0.09 95% CI -0.42, 0.23 $P=0.57$).

The finding that athletes with prior hamstring injury display reduced changes in peak torque over a single preseason is based on one HQ study (65) and is therefore unaffected by the sensitivity analysis. The outcomes provided by Mendiguchia et al (106) are of sufficient quality to be included in this sensitivity analysis. They provide very limited evidence of medium reductions in peak power, peak horizontal and force and a medium increase in time to reach 5m during a maximal sprints when comparisons are made between previously injured and uninjured subjects at return to play. All other papers are excluded from this sensitivity analysis owing to low methodological quality.

3.3.7 Subgroup analysis

3.3.7.1 Elite athletes

When data analysis is confined to research on elite athletes, there is moderate evidence indicating no change in concentric knee flexor peak torque (60°/second) in previously injured elite athletes (SMD -0.41 95% CI -1.09, 0.26). There is very limited evidence based on a single paper indicating moderate reductions in this value are observed at a 30°/second contraction velocity (SMD -1.12 95% CI -2.08, -0.16). Limited evidence also indicates no reduction in eccentric knee flexor peak torque in previously injured elite athletes (SMD -0.21 CI -0.79, 0.37 P= 0.48). Single paper evidence provides very limited evidence of large reductions in eccentric knee flexor peak torque in previously injured elite athletes at both 30°/second (SMD -1.59 95% CI -2.63, -0.55) and 230°/second (SMD -1.84 95% CI -2.93, -0.76) contraction velocities. Only one LQ study report angle of peak torque (APT) measures on elite athletes and provides very limited evidence of a large increase in APT during concentric knee flexor contractions (SMD 1.54 CI 0.62, 2.46). No analysis of APT during eccentric knee flexor contractions in elite athletes is available in the literature. Analysis of isokinetic ratio outcomes within elite athletes indicates moderate evidence of no reduction in the conventional H:Q ratio (60°:60°/second) between previously injured and uninjured conditions (SMD -0.21 CI -0.57, 0.14 P=0.24). Measures of the functional H:Q ratio indicates moderate evidence of a medium reduction in previously injured athletes at a 30°:240°/second contraction velocity (SMD -0.78 CI -1.2, -0.37 P= 0.0002). Furthermore, there is moderate evidence of a small reduction at the 60°:60°/second velocity (SMD -0.50 CI -0.99, -0.02 P=0.04). The NordBord outcomes are all based on elite athletes only and therefore remain unchanged when compared to the full analysis. The other studies which used an elite athlete population are Medighuchia et al (106) (radar based sprint outcomes),

Brughelli et al (102) (centre of mass forces and sprint contact times), Barreira et al (101) (vertical and horizontal sprint forces) and Daly et al (103) (sprint kinematics and EMG ratios). They provide very limited evidence as described in the full analysis for an elite population.

3.3.7.2 Confirmed exposure to evidence based rehabilitation

A sub-analysis was completed with subjects limited to those where a confirmed, evidence based progression had been completed. This was only present in seven papers (43, 65, 69-71, 84, 120). There is moderate evidence indicating no change in concentric knee flexor peak torque (60°/second contraction velocity) in this subgroup (SMD -0.14 CI -0.60, 0.31 P=0.54). Only single paper (very limited) evidence is available for eccentric knee flexor peak torque measures, which indicates no alteration in this subgroup for this outcome. Again, only single paper (very limited) evidence indicates no difference in angle of peak torque measures in previously injured athletes. The conventional H:Q ratio was not tested in athletes who had completed a stated rehabilitation programme. However, there is very limited evidence (84) indicating a medium reduction in functional H:Q ratio in this subgroup (SMD -1.1 CI -1.7, -0.50) at 30°/second:240°/second contraction velocity when measured at return to play. This deficit appears to resolve at 6 months post return to play. Moderate evidence indicates no difference in NordBord measured inter-limb peak torque in previously injured athletes who had completed a rehabilitation programme (SMD -0.34, 95% CI -0.83, 0.15 P= 0.17). However, there is moderate evidence of a small reduction in NordBord peak torque measures when the previously injured limb is compared to the left leg (but not the right) of a control group (Left leg: SMD -0.53 CI -0.95, -0.12 P= 0.01). Opar et al provide very limited evidence of persistent EMG deficits in spite of rehabilitation in two separate studies (69, 70). These outcomes are unchanged when compared to the full analysis and suggest reductions in peak and integrated EMG activity in the biceps femoris during eccentric isokinetic loading. There are no other outcomes tested in a population that had completed a defined rehabilitation programme.

3.4 Discussion

This review provides evidence that specific and persistent biomechanical deficits exist in athletes who have returned to sport following hamstring injury. However, the current evidence base lacks high quality research. Nevertheless, the persistence of post-injury biomechanical deficits indicated by this review suggests potential failures in current hamstring injury management strategies. This review illustrates key outcomes which require close monitoring during rehabilitation. Further high quality research is required confirm these findings and to assess if detected deficits respond to targeted rehabilitation. Furthermore, research is required to ascertain if a resolution of detected biomechanical deficits is associated with reduced re-injury rates. Such information may highlight outcome measures which offer valuable screening and rehabilitation tracking applications.

3.4.1 Evidence of deficits in force production:

3.4.1.1 Isokinetic dynamometry.

The majority the literature suggests that there is no significant difference in isokinetic performance between previously injured and uninjured athletes after return to play. There are some exceptions, with moderate evidence of altered eccentric knee flexor peak torque at 30°/second (medium reduction), an altered conventional H:Q ratio using 60°:60°/second velocities (small reduction), and an altered functional H:Q ratio using 30°:240°/second velocities (medium reduction). The angle of peak torque has only been assessed in a limited number of lower quality studies. Consequently, only limited and very limited evidence indicates that this measure appears unaffected in previously injured subjects during concentric contractions. Very limited, single paper evidence suggests that this outcome is reduced (medium SMD) during a 30°/second eccentric load. The lack of high quality evidence means that many isokinetic outcomes are not assessable by sensitivity analysis highlighting the need for further research with an improved methodological approach.

Overall, these findings suggest that slow, eccentric contractions may be more sensitive in detecting deficits in force production in subjects with previous hamstring injury. There are a number of reasons that this may be so. Modern rehabilitation approaches, as described by Heiderscheit et al (60) and Silder et al (63), are focussed on restoring muscle function and postural control at higher contraction velocities during sport specific movement

patterns. This would appear justified given the current appreciation that sprint related hamstring injury occurs during high speed movements and such rehabilitation techniques are therefore both more sport and injury mechanism specific (25, 85, 87). However, this approach to rehabilitation may also neglect heavy eccentric loading at slower contraction velocities. There is evidence to indicate that eccentric contractions are associated with greater torque outputs when compared to concentric contractions (75). There is also evidence that high load eccentric contractions may recruit previously inhibited motor units thus inducing a near total recruitment of a muscle (122). In addition, eccentric loading requires greater cortical input and inhibition of antagonistic muscle activity (74, 75). Perhaps exposing previously injured athletes to slow, maximal eccentric loads may expose subtle deficits in previously injured subjects that are not detectable during higher velocity, submaximal and/or concentric type contractions.

3.4.1.2 NordBord

The NordBord™ is a novel device developed to measure force production at the ankles (and indirectly at the hamstrings) during a Nordic drop, a slow, high intensity, eccentric contraction of the hamstrings. There is moderate evidence of a small reduction in peak force on the previously injured limb when comparison is made with the opposite limb in previously injured subjects and a medium reduction versus the right limbs of control subjects. These finding is not supported by the sensitivity analysis and no significant differences were noted when meta-analysis is limited to subjects who had completed an evidence based rehabilitation protocol. The NordBord may be a simple alternative to isokinetic dynamometry in identifying and tracking post-injury deficits in knee flexor function. It appears that deficits measured using the NordBord may be less detectable, or may not exist, in those who have completed a defined rehabilitation protocol.

Nevertheless, this review indicates a need for further high quality studies with the NordBord. A validity study is also warranted, in order to determine the sensitivity of the NordBord versus more traditional isokinetic dynamometry, especially at slower contraction velocities. Notably, the Nordic drop is traditionally performed in a kneeling position with the hips in a neutral position (64). This is in contrast to isokinetic dynamometry, which is typically performed in sitting (i.e. with hips flexed). Therefore, direct comparisons regarding validity may be difficult as the hamstrings are operating at differing functional lengths during each test.

3.4.1.3 Forces during running

Terminal swing in high speed running is the phase where sprint related hamstring injury occurs (25, 51, 85, 87). However, evidence related to force during running in previously injured athletes is lacking. Studies which do explore such outcomes differ in their methods, thus further limiting interpretation and provide very limited evidence at best. Only one study (116) measured forces specifically during late swing and provided very limited evidence of no deficits in previously injured subjects in hip and knee moments and powers. Whole body peak horizontal force was reduced during sprinting in two studies using the contrasting methods of strain gauges and radar telemetry (102, 106). However another found no change in ground reaction forces on an instrumented treadmill (101). Whichever method is employed, further research is needed to truly determine the association between previous hamstring injury alterations in functional force production during running.

3.4.1.4 Hand held dynamometry

Only one study (43) employed hand held dynamometry as an outcome measure in previously injured subjects. Unfortunately, only differences between un-injured and injured limbs were reported and SMD analysis was not possible. Nevertheless, deficits in force production at 15° knee flexion were predictive of a future re-injury when multivariate regression analysis was employed. There is insufficient published data to determine the value of HHD in measuring alteration in function in previously injury athletes.

3.4.2 Evidence of deficits in movement

3.4.2.1 Temporal

Only three studies explored temporal aspects of movement in previously injured athletes. With differing methodologies, definite conclusions cannot be drawn via meta-analysis. Nevertheless, Mendiguchia et al (106) do provide limited evidence indicating reductions in early sprint acceleration evidenced by increased time to reach 5m in previously injured elite level footballers when measured at RTP. These footballers showed no deficits in time to reach 10m or 40m. Their rehabilitation methods were not outlined and it is therefore not known if such deficits would be present following sport specific/sprint focussed rehabilitation. Nevertheless, these deficits were resolved at the time of follow-up testing (~2 months post RTP) suggesting that this outcome measure may be sensitive in the early

rehabilitation phases only. The remaining studies both provide very limited evidence of no change in ground contact times between injured and uninjured limbs and controls during high speed running (102) and faster reaction speeds (-54ms on previously injured side) when asked to perform a double to single leg standing movement (109). Therefore, the clinical value of measuring temporal aspects of movement following hamstring injury remains unknown.

3.4.2.2 *Kinematics*

There is some very limited evidence suggesting that there is an association between prior injury and altered movement patterns. Daly et al (103) found asymmetrical movements at the pelvis and hip during late swing characterised by increased anterior pelvis tilt and hip flexion on the injured side, when comparison is made between previously injured Gaelic games athletes (hurlers) and their teammates. Furthermore, these authors also detected asymmetrical patterns at the time of initial foot contact at the knee, characterised by increased an increase in medial rotation on the previously injured side. These athletes were not exposed to a confirmed rehabilitation protocol but were deemed to be elite in their sport and had returned to full sporting participation. In contrast, Lee et al (116) explored inter-limb differences in previously injured subject, using the contralateral (i.e. uninjured) limb as the control comparator. These authors did find a significant difference characterised by decreased late swing hip flexion on the previously injured limb. Perhaps the differing methodologies may account for the contrasting results reported by each of these authors. Daly et al (103) quantified asymmetry across multiple time points using a bootstrapping technique to make comparison between uninjured and previously injured subjects. In contrast, Lee et al (116) used a more typical approach and compared mean values within phases of the gait cycle and made comparison between the limbs of previously injured subjects. Brughelli et al (102) employed the most simple movement analysis by examining centre of mass displacement using an external rig. They provide very limited evidence for no difference between limbs or versus controls in previously injured athletes. Silder et al (119) provide limited evidence of no difference in biceps femoris musculotendinous length in previously injured limbs. These authors used musculotendinous modelling that incorporated extrapolated musculotendinous lengths from kinematic data making comparison between the limbs of previously injured subjects who were not classified as elite nor had they undertaken any standardised rehabilitation.

When the three studies exploring running kinematics (or outcomes derived from kinematics) are considered together, it is apparent that differences appear more readily when comparison is made with well-matched uninjured control subjects. Nevertheless, further research is required to establish if persistent changes in movement patterns during running exist in previously injured athletes.

3.4.3 Evidence of deficits using EMG

In all but one study, significant changes in hamstring EMG activity was noted in previously injured subjects. Although differences exist in their approach to signal processing, each of these studies found that previously injured hamstrings displayed reduced amplitude activity (or reduced activity relative to other paired muscles) versus uninjured comparators. Two studies (103, 108) found that these alterations were most evident when the muscle was at its greatest functional length and the time of greatest biceps femoris injury risk (25, 85, 87).

Two studies explore EMG outcomes during running. Amplitude outcomes are typically presented as a percentage of a reference value (i.e. are normalised) to account for inter-subject variation in signal amplitude. However, there is currently no widely accepted guidance pertaining to the reporting of muscle activity, where suspected neuromuscular dysfunction may be present. Expressing deficient muscle activation levels as a percentage of a deficient comparator (whether maximal or average) may reduce (or negate) real differences. Daly et al (103) attempt to deal with this dilemma by avoiding normalisation, instead expressing their results as activation ratios between functionally relevant pairings. They provide very limited evidence indicating reduced activation ratios and possible impaired biceps femoris activity during terminal swing in high speed running in previously injured athletes. Silder et al (119) use a more established approach and express EMG amplitudes as a percentage of mean activity across the entire gait cycle. They further reduce their data to average normalised activity across four phases of running gait finding no differences in activation between the previously injured and uninjured limb in previously injured athletes from a wide range of sports. Furthermore, they report no difference in activation timings between limbs.

Three studies explore EMG outcomes during isokinetic dynamometry. All three employed typical normalisation methods with Opar et al (69) expressing EMG amplitudes as a

percentage of peak activity, Opar et al (70) expressing integrated EMG values as a percentage of peak activity and Sole et al (108) reporting root mean squared values expressed as a percentage of peak activity. All three report reduced hamstring activity in previously injured limbs. Finally, Sole et al (108) report significant reductions in normalised RMS activity during eccentric contraction at 60°/second. Of note is that post-hoc analysis suggested that these findings were most obvious in the functionally important outer range.

Although differing methodologies were employed, there is a consistent indication (albeit with very limited evidence) of detectable deficits in hamstring muscle activity during eccentric isokinetic loading. There is therefore some value in exploring such outcomes in further studies of high methodological quality.

3.4.4 Sensitivity analysis

When low quality studies are omitted from the meta-analysis, it is notable that only single paper (i.e. very limited) evidence exists in all outcomes with the exception of the conventional H:Q ratio (at 60°/second) and some of the NordBord results. In these cases, sensitivity analysis provides findings which are insignificant and in contrast to the full meta-analysis. Therefore, the current evidence base is notably lacking in terms of methodological quality. Further high quality research is need to firmly establish the biomechanical characteristics of individuals with prior hamstring injury.

3.4.5 Subgroup analysis

3.4.5.1 Elite athletes

This subgroup analysis was completed to ensure generalisability of the findings to an elite population. In spite of probable exposure to medical and rehabilitation supports, this review indicates that elite athletes display clear signs of hamstring associated deficits following return to play after hamstring injury. In particular, there is moderate evidence of medium reductions the functional H:Q ratio when a 30°:240°/second contraction velocity combination is employed. Deficits in maximum force between previously injured limbs and contralateral limbs, as well as the limbs of control subjects is confirmed in elite athletes via the use of a NordBord (moderate evidence). Very limited evidence indicates reduced horizontal force production at the centre of mass during high speed treadmill running (102), reduced peak power and peak horizontal force production at RTP during maximal sprinting (106) and impaired acceleration ability over 5m at RTP but not at ~2 month

follow-up (106). There is also very limited evidence indicating increased asymmetries in previously injured Gaelic games athletes characterised by increased late swing anterior pelvic tilt and hip flexion as well as initial contact medial knee rotation (103). EMG ratios are also reduced (limited evidence) during terminal swing between the biceps femoris and ipsilateral gluteus maximus, erector spinae and external obliques suggesting inhibited activation of the biceps femoris (103).

3.4.5.2 Exposure to evidence based rehabilitation.

Only seven of the 28 papers included imposed controls on the rehabilitation undertaken with previously injured subjects. These programmes were characterised by progressive loading, a focus on eccentric load and sports specific elements. Nevertheless, some deficits appear to persist in this population, perhaps suggesting an inadequacy of current rehabilitation approaches. Single paper (i.e. very limited) evidence indicates deficits in both concentric and eccentric peak torque as well as a shifting of the angle of peak torque to shorter hamstring lengths during eccentric contractions. When limited to this sub-group, there is no significant difference in NordBord outcomes, a finding which is in contrast to the full analysis. Furthermore, only one paper measured the functional H:Q ratio in this population with no significant change noted.

3.4.6 Clinical implications

Prior hamstring injury remains the single biggest risk factor for future hamstring injury across sprint related sport (2, 6, 7, 11, 16, 67, 82, 123). The persistent increased re-injury risk following an initial hamstring injury indicates shortcomings in current rehabilitation strategies. In recent years, the focus of rehabilitation following hamstring injury has shifted to a combination of eccentric loading, postural agility and trunk stability work and sport specific training including progressive exposure to high speed running drills (60). The emergence of the Nordic loading protocol (64), the Postural Agility and Trunk Stability (PATS) protocol (62), progressive sprinting protocols (63) and combinations of the above (60) as well as the associated evidence indicating reduced injury risk following their implementation is highly positive. Nevertheless, we continue to lack a clear understanding of the nature of the deficits which are resistant to rehabilitation following injury. Consequently, we lack the ability to adequately screen athletes for such deficits.

This review provides clear evidence of persistent biomechanical deficits associated with previous hamstring injury. This review indicates that these deficits appear to be detectable with isokinetic dynamometry, especially eccentric peak torque values at slower velocities and reduction in the functional H:Q ratios following return to play. There is a tentative indication that slower velocities and eccentric contractions appear most sensitive in detecting such deficits. The NordBord may offer an alternative method to detect force production deficits in previously injured athletes with clear indications of asymmetries in previously injured elite athletes.

This review indicates that the evidence to indicate prior injury associated deficits in movement and/or EMG is less established. Very limited evidence indicates deficits in force production, power and acceleration during running, increased asymmetries in running at the pelvis, hip and knee and altered EMG activity in the biceps femoris during running during isokinetic testing.

3.4.7 Methodological limitations and considerations for future research

A large number of studies reported isokinetic outcomes. When suitable for meta-analysis high levels of homogeneity were found between differing studies. This ensures an enhanced level of confidence in the evidence reported. There was significant variability in methodologies used to analyse movement and EMG outcomes in included articles. This meant that data pooling was not possible or justifiable. The high level of variability in methodology perhaps highlights the challenges which exist in measuring functional movement and EMG in previously injured participants, especially during high speed running.

In several of the isokinetic papers, data was reported for several outcomes using the same subjects. For instance, a number of papers report peak torque values for more than one contraction velocity. In this review, we included each contraction velocity as a separate outcome regardless of the source. We considered each velocity as a difference test and therefore feel that we do not present underpowered data. In two papers (117, 118) both relative (Nm/kg) and absolute (Nm) peak torque outcomes are presented for each contraction velocity. In this case only the relative outcomes were included in the meta-analysis to avoid overpowering.

Only three studies indicate that the participant numbers are sufficiently powered according to sample size calculations. Although this may mean that individual studies are under-powered, increasing the risk of reporting error, pooling of data for outcome where possible, negates the risk of such error in this review.

No studies have examined the prospective effect of hamstring injury on biomechanics. All injured participants were recruited after sustaining a hamstring injury and therefore only associations between a history of injury and the presence of biomechanical deficits can be made. Determining if such deficits were present in advance of injury is not possible without a prospective analysis. All but three studies fail to report incident cases of injury during the period of follow-up and where reported this was limited to the previously injured group only. In the absence of prospective data, comparisons can be made between injured and uninjured limbs as well as between previously injured and uninjured control subjects. However, it is imperative if employing a case-control design, that careful matching of controls must be completed to ensure to effects of confounding factors are kept to a minimum. All except two studies fail to consider environmental confounders when they carried out the analysis on subjects. The majority of studies also fail to consider the potential effects of individual confounders when presenting their results. Observer blinding was not reported in all included articles increasing the risk of observer bias in individual studies. The retrospective nature of all included studies did not easily facilitate the recording of baseline characteristics of subjects in their pre-injured condition. This again highlights that any deficits measured were associated with, rather than caused by, the injury.

Although many of the included articles report severity of injury and time since injury, none adjust their analysis to account for variability in outcomes secondary to either of these factors. However, it is also acknowledged that the majority of studies simply did not have sufficient subject numbers to enable such subgroup analysis. Small subject numbers alongside a lack of sample size calculations mean that it is not possible to determine the generalisability of outcomes either within the groups being testing or the wider sporting population.

Future research should therefore seek to establish if prior hamstring injury is associated with altered movement patterns and/or surface EMG outcomes, particularly during high

speed running. In order to achieve the latter, a wider consensus on how one should handle EMG data in potentially injured or dysfunctional muscle is required. Furthermore, although isokinetic peak torque and the functional H:Q ratio appear to be reduced in previously injured athletes, future studies should see to explore these outcomes in subgroups which consider time since injury, severity and nature of injury.

3.5 Conclusion

Athletes who have returned to sport display detectable alterations in force generation capacity, movement patterns and EMG activity after they have returned to sport. In many cases these deficits appear present even after athletes have completed a rehabilitation protocol according to current best practice. Some deficits also appear to be present in an elite athlete population. Force generation deficits are noted most during slower velocity and eccentric type contractions. These areas are key rehabilitation targets. Further research is required to quantify if successfully addressing these issues lead to reduced re-injury risk.

4 Materials and methods

4.1 Introduction

The following chapter describes the development of the methodologies designed to meet the stated objectives of each of the four experimental studies included in this thesis as detailed in Chapter 2.

4.2 Participants

4.2.1 *Ethical procedures*

Each experimental component of this thesis involved the testing of human participants. At all stages, this research was conducted in accordance with the Declaration of Helsinki (124). As such, each experimental study protocol was subject to prior independent review and approval by a university ethics committee in advance of commencing recruitment. Independent ethical approval was obtained via both Queen Mary University of London and University College Dublin human research ethics committees for the study outlined in Chapter 5 (see Appendix 9.4 for documentation). Ethical approval for the studies outlined in Chapters 6 and 7 were obtained via review by the Queen Mary University of London human research ethics committee (see Appendix 9.4.2, 9.4.3).

The principles of the Declaration of Helsinki (124) were upheld as follows.

- *Informed consent*

All participants volunteered to take part in these research projects. In advance of providing written consent to take part, all subjects were provided with a participant information sheet which provided a clear overview of the study aims, methods, details of researchers, anticipate risks and benefits and follow up provisions in plain language written format. Subjects were also advised of their right to refuse or subsequently withdraw from the research process at any stage without penalty. Following verbal clarification that the subject understood the participant information sheet they were invited to provide written confirmation of their willingness to take part by completing a written consent form. Subjects were provided with the opportunity to review the study information sheet and consent form without influence from anyone with who they had a prior dependent

relationship (e.g. club doctor, club physiotherapist, coach) in order to avoid undue duress to take part. All subjects were deemed capable of making an informed decision and were not members of a vulnerable group.

- *Risks, burdens and benefits*

An outline of any risk involved in taking part in this research was provided to potential participants within the information sheet. Particular emphasis was made to the risk of reinjury, but care was taken to ensure participants remained symptom and pain free for the duration of testing this reducing the risk of injury. Furthermore, participants were recruited after return to sport for both the running study (Chapter 5) and the Nordic loading study (Chapter 6). The testing procedures were designed to be within the safe capabilities of a subject who had returned to full participation in sport.

- *Privacy and confidentiality*

In order to ensure anonymity, subjects were allocated a five-digit code upon recruitment to a study. Information linking this code to the participant's name was recorded and stored in a locked cabinet with access limited to the PhD candidate. All data collected from subjects was subsequently stored on a password protected harddrive using the subject code only. Subjects' physical privacy was maintained during the data collection setup phase through the use of screens within the human performance laboratory environment.

- *Follow-up access*

All subjects were provided with the phone number and email address of the principle researcher (primary PhD supervisor), the PhD candidate and the research ethics secretary. They were invited to contact any of these individuals if they had any questions regarding the study or the manner in which it was conducted respectively.

It is noted that the ethical documentation used for the studies in this thesis was incorporated as a teaching tool for undergraduate and Masters students in the QMUL Sports and Exercise Medicine faculty. This documentation became the *pro forma* for a significant number of subsequent applications.

4.2.2 *Sample Size*

The methods employed in this thesis are quite novel, both in terms of analytical processes and methodological approach. It was not possible therefore, to prospectively calculate study sample sizes to ensure sufficient powering. Therefore, we instead used a retrospective analysis of effect sizes to examine statistical power in both observational case control studies (Chapters 5 and 6) (125). Cohen's d was calculated as the following: the difference in means between groups was determined and divided by the standard deviation of the data in the control group. The control group was selected as a representative example of a normal population. In the running study (Chapter 6), data was examined across 101 timepoints representing the entire stride. Calculations were made at each point for both the kinematic and EMG ratio data. In the Nordic loading data, the estimated marginal means were calculated using SPSS (IBM Inc., Armonk, NY, USA, Version 22) software and this data was used to calculate Cohen's d . The effect size was described using the descriptions provided by Cohen (125), namely, small ($d = 0.2$), medium ($d = 0.5$), and large ($d = 0.8$).

4.2.3 *Recruitment*

Subjects were recruited by utilising pre-existing and newly fostered links to various sporting clubs and organisations in both Ireland and the south-east of England. The author had pre-existing links to elite Gaelic Games clubs in Dublin having worked as a physiotherapist with a number of teams in the region prior to commencing this PhD. This provided access to a large population of elite level Gaelic games athletes for the running study outlined in Chapter 5. Potential participants were provided with written information in poster form regarding the study and the author attended training sessions to provide additional information as necessary. This phase of data collection occurred in University College Dublin which was convenient for participants.

Subject recruitment for the later observational study on low density, high surface area EMG and the case studies was via new and pre-existing links with UK based sporting organisations. Throughout the PhD process, the author supervised a number of undergraduate and postgraduate students from the Sports and Exercise Medicine academic group, who assisted with aspects of data collection as part of their own research projects. Several of these students had pre-existing links to university level football teams, as well as

professional football clubs in the London region. Their prior relationships enabled access to these groups for recruitment. In addition, dissemination of early findings via publication, conference attendance and educational lectures by the author to club medical teams led to direct approaches from individual working in elite sport who wished to be involved in this research. Finally, the author also fostered new personal links with individuals working in elite sport in the UK, and these relationships enabled further recruitment.

Throughout the PhD process it was noted that successful recruitment was dependent on forming a personal relationship with individuals who worked alongside or had close links to potential study participants. Although direct approaches via email to club officials and poster recruitment were attempted, these methods rarely resulted in successful recruitment of participants. Where recruitment was successful via such methods (2-3 participants in total), the anticipated recruitment of participant colleagues via snowballing did not materialise. For this reason, effort was diverted away from establishing new contacts and pre-existing relationships were fostered instead. In spite of the range of direct and indirect relationships which emerged during the PhD process, it is acknowledged that recruitment remained challenging for the duration of the various data collection phases.

4.2.4 Inclusion and exclusion criteria

All recruited subjects were assessed to ensure they met specific and predefined inclusion and exclusion criteria. This was based on descriptors provided in previously published research (117, 118). Subjects in the previously injured group described a subjective history consistent with sprint related hamstring strain. Specifically, previously injured participants reported: (a) sudden onset of posterior thigh pain which occurred without contact and during high speed running, (b) pain which was localised to the hamstring region, (c) active hamstring contraction and running causing discomfort for a minimum of 48 hours following injury, (d) an inability to participate in sport for at least 48 hours following injury. As a secondary means of injury confirmation, participants were questioned regarding the presence of bruising following injury, whether they underwent contemporaneous assessment by a registered physiotherapist/medical practitioner and/or confirmatory imaging. The time since injury was recorded. All subjects had returned to full activity in sport.

The time since injury was limited to a maximum of 24 months in the running study (Chapter 5) but was extended to 36 months for the Nordic loading study (Chapter 6). This was based on the growing recognition that post-injury deficits appear to persist for a long period post-injury (61) and long term follow-up was determined necessary to measure and long lasting dysfunction.

Subjects were excluded from the observational studies if they displayed signs of weakness on manual muscle testing (126), had signs of neurodynamic dysfunction based on a positive slump test, or articular dysfunction at the sacroiliac or lumbar spine region using manual testing and movement screening (127). All clinical screening was carried out by an experienced musculoskeletal physiotherapist.

It is acknowledged that the inclusion criteria are based on subjective reporting only. This method is consistent with many previously published methods but is vulnerable to subject recall bias. We sought to reduce the risk of bias by collecting secondary data on confirmation of diagnosis via professional clinical assessment and/or imaging.

4.3 Analysis of running biomechanics

The following section provides an overview of the method development for the running analysis employed in Chapter 5 of this thesis. Detailed descriptions of the outcome measures employed are provided. Furthermore, the methodological approach to data extraction and analysis is described from development phases through to final implementation. The aim, objectives and hypothesis for the associated study is outlined in section 2.1.2 and 2.2.2. The associated matlab code used for data processing is located in section 9.2.1.

4.3.1 *Measuring kinematics*

A three dimensional analysis of running was completed by measuring lower limb and pelvic movement using a motion capture system. The CODAmotion system (CX-1 units and software, Charnwood Dynamics Ltd., Leicestershire, UK) is an infra-red active marker, motion capture system which is available in both University College Dublin and Queen Mary University of London motion capture laboratories – both centres being used during the data collection phase of this PhD. This system uses information gathered from light emitting diode (LED) markers placed upon a subject's body, to calculate their movements

within a laboratory reference frame. The resolution of this system is 0.03mm in the lateral direction and 0.3mm in distance. Data is collected at 200hz when using the standard marker arrangement.

4.3.1.1 Calibration

The calibration process for the CODAmotion system establishes a Cartesian reference frame (the laboratory frame) in a user defined collection volume prior to data collection. Scanner units (cameras) are placed around the capture area and are orientated towards the centre of the space. An origin point is selected within the data capture space and a Cartesian frame is established about this point. This is achieved through the placement of LED markers at the origin point as well as along two of three axes (x, y, z) from this point. Typically, a second marker is placed along the x axis and a third a point perpendicular to the primary axis to defined the y direction. The calibration process establishes the location of the origin (x_0 , y_0 , z_0) in relation the each of the scanner units, as well as the orientation of the x and y axes. Having established a plane from the x and y axes, the orientation of the z axis is defined as being orthogonal to the defined plane. During data capture, the location of each marker is measured in relation to the origin and is described with x, y and z coordinates measured in millimetres. In order to maximise visibility with the data capture area, three or four scanner units were positioned on tripods encircling the area. It was imperative that these scanner units remained static following calibration, as any repositioning would invalidate the location of the origin and thus corrupt the data. Therefore, if any repositioning of a scanner unit occurred, a recalibration was completed.

4.3.1.2 Measuring human movement

The CODAmotion system and analysis approach utilises a modified version of the Helen-Hayes marker protocol (128) to capture lower limb and pelvic movement patterns. The Helen Hayes protocol allows for the calculation of joint movement about 3 planes of motion (sagittal, coronal and transverse). The modified Helen Hayes protocol used with the CODAmotion systems is based on 22 markers which are placed on a subjects body as outlined in Figure 14. The markers are positioned predominantly on bony landmarks and provide data for segmental analysis of lower limb and pelvic movement. The lower limbs are subdivided into seven segments (pelvis, left and right thigh, left and right shank, left and right foot), each having a minimum of three associated markers. Tracking of these

markers within the capture space enables the calculation of individual segmental movements using an embedded vector basis (EVB) approach. An EVB is a local coordinate system for each segment. For instance, the thigh segment coordinate system is as follows: the z axis is formed by a line joining the knee and hip joint centres, the x axis is formed by the line of the femoral wand positioned perpendicular to the transfemoral axis, and the y axis is perpendicular to the x and z axes. At the shank and thigh an instrumented wand is used to establish the y axis and enable calculation of transverse plane motion. Joint centres for the ankle and knee are based on geometric measurements of joint widths and the placement of the knee and ankle markers respectively. The hip joint centre is calculated from the anterior pelvic markers using predetermined offsets based on the work of Bell *et al* (129).

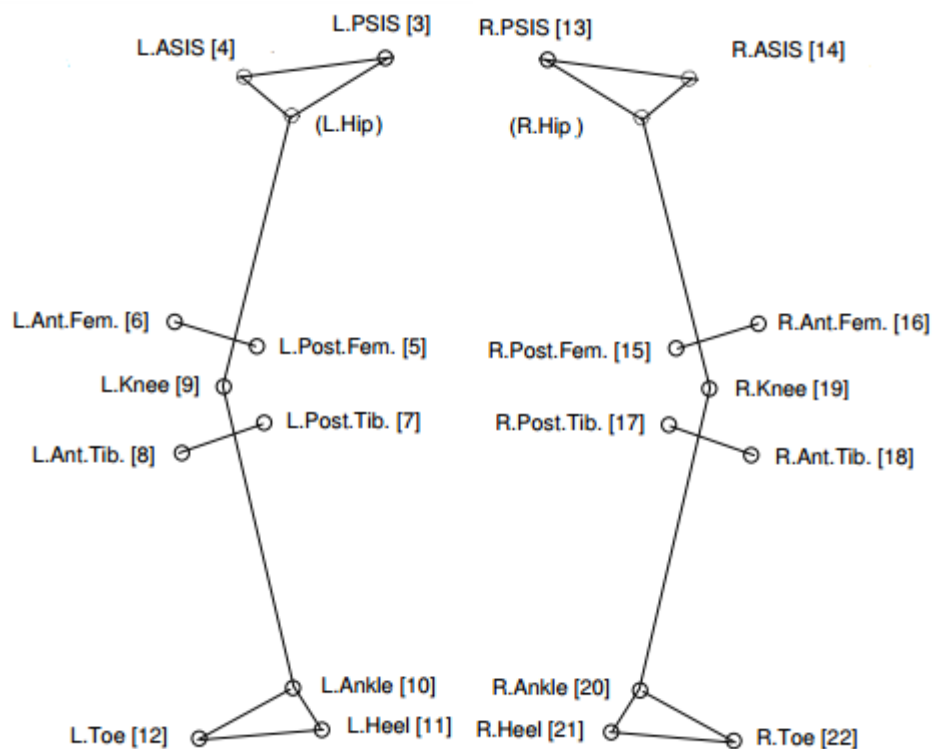


Figure 14: Modified Helen-Hayes marker placement protocol for CODAmotion system (128). L=Left, R=Right, PSIS=Posterior superior iliac spine, ASIS= anterior superior iliac spine, Ant=anterior, Post=posterior, Fem=femur, Tib= tibia. Hip joint centres are included for illustrative purposes only and are not marked in the model.

Once the EVB segmental rotations and joint centre locations are known, the CODAmotion software calculates the Euler angles occurring at each joint by considering the movements of the proximal and distal segments about the joint in question. In the case of the pelvis

and foot, rotations are considered with respect to the laboratory coordinate system. The foot rotations are not reported within this thesis.

This system and the methods used to compute joint movement have been found to be a valid and reliable measure of movement in human gait (128). The accuracy of this approach is however dependent on a number of factors. The positioning of the femoral and tibial wands is prone to user error. The markers located on these wands equates to the x axis of their respective segment EVB, for example see L.Ant.Fem, L.Post.Fem in Figure 14. Therefore, any inaccuracy in the positioning of these wands would lead to measurement error resulting in erroneous interactions between the transverse and coronal plane data at the hip, knee and knee. The effects would manifest as incorrect measures of internal/external rotation at these joints as well as incorrect measures of hip abduction/adduction, knee valgus/varus and ankle supination/pronation. Particular care was therefore taken to ensure that the femoral and tibial wands were positioned along a line perpendicular to the transverse femoral and tibial axes respectively using the methods described by Monaghan *et al* (128). Great care was also taken to ensure accurate placement of the knee, ankle and anterior superior spine markers, as any misplacement would also lead to measurement error related to the calculation of joint centres. These markers were firmly attached to the correct anatomical landmarks and further supported with adhesive surgical tape prior to data collection. The main data collector undertook extensive training in the user of the CODAmotion system under the supervision of highly experienced research staff. Reliability of the main data collector was checked and compared to the literature where possible and modifications in approach were made to ensure accuracy of data collection. Furthermore, the main data collector has over ten years experience working as a musculoskeletal physiotherapist and therefore has a detailed knowledge of human anatomy and advanced palpation skills.

4.3.2 *Measuring EMG*

Surface EMG is a non-invasive and indirect measure of muscle activity commonly used in human performance analysis (130, 131). A muscle contraction is initiated by the arrival of an action potential along the alpha motor neurone at the motor end plate. This stimulus triggers a response in the sarcolemma, the membrane which surrounds the muscle. An influx of sodium ions into the sarcoplasm causes a depolarisation of the sarcoplasm and

triggers the release of calcium into the intracellular space. The calcium interacts with molecules attached to the sarcomere and triggers an excitation-coupling effect across the actin-myosin cross bridges, resulting in a muscle contraction. The sarcoplasm is rapidly repolarised. The depolarisation and repolarisation of the sarcoplasm propagates throughout the muscle fibre via the sarcolemma and its arrangement of t-tubules and reticula (131-134).

As the action potential propagates along the muscle fibre away from the end plate, a depolarisation wave is created by the depolarising component travelling forwards with the repolarising component behind. With bipolar EMG (us used in Chapter 5), the potential difference between the advancing depolarisation and the chasing repolarisation is measured. All electrical activity within the range of the sensors are detected and summated (130, 133). The amplitude and density of this signal is dependent on the number and firing frequency of the underlying activated motor units and other electrical signals within the skin. In addition, subcutaneous tissues act as a low pass filter to the action potential signal. The surface EMG signal is therefore a composite signal of multiple motor unit action potentials the mean activity of which is equal to zero (130). Motor unit recruitment in skeletal muscle approximates to a sequential sequence known as the Henneman principle with higher force generation involving the recruitment of larger amplitude motor units and faster firing frequency muscle fibres (135). The result is an increase in amplitude and density of the signal. Therefore, the raw surface EMG can be considered as representative of underlying muscle activation but it does not directly equate to the firing frequency or amplitude characteristics at the level of the motor units.

A number of factors can adversely influence the accuracy and repeatability of the surface EMG signal. These factors are described, and the measures taken to minimise their influence in these research projects are outlined in the following sections.

4.3.2.1 Crosstalk

The quality and accuracy of the EMG signal can be adversely affected by EMG activity in muscles adjacent to the target muscle. Other physiological signal can also be detected by the surface electrodes such as electrocardiogram (ECG). We were careful to position the electrodes over the muscle belly of the target muscle. Careful palpation of muscle borders and testing of contraction using resisted exercises were used to confirm accuracy as per

recommended guidelines (136). Specific sensor locations for each of the muscles tested is outlined in Table 6.

Muscle	Position of sensor
Biceps Femoris	50% along a line joining the ischial tuberosity and lateral epicondyle of the tibia.
Gluteus Maximus	50% along a line joining the greater trochanter and S1. The most prominent point of the muscle
Erector Spinae	Mark L3 spinous process and measure 3cms lateral this point, ensuring that the position is still within the boundary of the muscle
Rectus femoris	50% along a line joining the anterior superior iliac spine and the superior patella
External Oblique	15cm lateral to the umbilicus ensure you are not over the lateral aspect of rectus abdominus

Table 6: Location of sEMG sensors for running study (136).

Interference from ambient signals such as poorly earthed electrical cabling and treadmills can also interfere with the quality of the EMG signal. The EMG electrode used for the running data in the thesis were specifically designed to minimise many of the potential interference from these source. The Motion Lab Systems EMG electrode (see Figure 15) in was used during the running study outlined in Chapter 5. This electrode is specifically designed to give accurate measurements during challenging activities such as treadmill running.



Figure 15: Motion Lab Systems Preamplifier MA-411 surface EMG electrode used in the running study in this thesis.

The electrode is comprised of two 13mm stainless steel sensing disc with a 17mm inter-electrode distance. The two sensing disc are interspersed with a ground electrode. The positioning of the ground adjacent to each pair of sensing discs is suggested to make the system highly resistant to ambient electrical noise (137).

4.3.2.2 Motion of the sensor

Motion artefacts are low frequency, often high amplitude interference on the EMG signal which results from movement of the sensor electrode on the skin of the participant. In addition, excessive cable movement can interfere with signal quality (130). The motion Lab Systems electrodes are preamplified at the patient end in order to reduce interference from cable movement. In addition, the participants skin was prepared by shaving and cleaning in line with SENIAM guidelines (136), to ensure maximal adherence of the sensors. Each sensor was taped in place with surgical tape (Meditrans, Fleming Medical, Ireland) to minimise any movement. The signal quality was continually inspected for any evidence of motion artefact and sensors were reinforced with more tape as necessary.

4.3.2.3 Impedance

Cleaning of the sensors and skin with an alcohol wipe as well as abrading of the skin with fine sand paper to remove any dead skin ensured minimal impedance of the EMG signal. This process was as per SENIAM recommendations (136).

4.3.3 Synchronisation

Data for the running study in Chapter 5 was collected using two separate systems. EMG data was collected using Motion Lab Systems software, while kinematic data was collected with the CODAmotion analysis software on a separate PC. Accurate synchronisation was ensured through the use of a digital trigger (HLT100 INISO, BIOPAC Systems LTD., CA. USA). which connected both systems. The EMG software was set to begin data collection on receipt of this digital signal which was triggered by the onset of data capture on the kinematic software.

4.3.4 Treadmill running

Given the restricted data capture space associated with 3D motion capture and the fact that subjects were tethered by the EMG leads, we elected to measure running on a treadmill. The treadmill offers some advantages when conducting kinematic analysis of running. Data related to multiple strides can be captured over a single trial. Gait data is somewhat variable by nature (138). Averaging across ten or more strides is recommended to improve the reliability of kinematic outcomes (128). A ten second data capture window with subjects running at 20km/h typically produces between 14 and 20 strides for analysis.

However, motion capture and EMG measuring during treadmill running also presents some significant challenges. The high impacts associated with high speed running can lead to motion artefact in the EMG record and repositioning of motion capture markers. We ensured that we minimised these effects through the application of a layer of flexible transparent surgical tape over all EMG electrodes and motion capture diode and batteries. Any loose wiring was also lifted and secured at chest height to avoid creating issues with motion capture marker visibility. Furthermore, the treadmill which we used (Trimaster 2000) did not have side arms. The CODAmotion scanner units were positioned behind and to the side of the participant to ensure maximal visibility.

It is accepted that treadmill sprinting is not an exact replicate of overground sprinting. The interaction between the athlete and the belt is different to that between the athlete and the ground. In particular, there is a limited body of research indicating alterations in gait parameters as well as kinematics when comparison is made between overground and treadmill running. Decreased stride times (0.02-0.03 s) and stride lengths (0.11- 0.14 m) are noted when running on a treadmill (139, 140) but the magnitude of difference is small and likely to be of limited clinical relevance. Furthermore, some kinematic differences are also noted between running conditions. Reduced stance phase anterior pelvic tilt ($\sim 2^\circ$) (141) and a 0.9° increase in transverse plane pelvic movement are reported (140). Hip flexion at initial contact appears to be reduced in treadmill running (-5.6 to -12°) (141, 142) but maximum hip extension appears unchanged between conditions (140). There is inconsistent evidence of small changes in movements in the coronal and transverse plane at the hip, with very small differences (less than 1.1°) noted between treadmill and overground conditions (140-143). At the knee, only small changes are noted across all planes, the largest being in the sagittal plane when treadmill running is reported to be associated with significantly ($p < 0.001$) reduced peak knee flexion through early swing (-6.6°) and increased terminal swing extension ($p < 0.001$; 1.9°) (140). Again the magnitude of these changes are small. Finally at the ankle tends to be more plantarflexed at initial contact during treadmill running (max 4.5°) (142, 143). EMG timing and amplitudes appear consistent between both conditions (144). Although small differences in kinematics exist between treadmill and overground running, the group comparisons in Chapter 5 were made with all subjects running on a treadmill. The results are therefore limited to

observations of treadmill running and some caution should be used when generalising to the overground running condition.

Finally, as hamstring injury is thought to occur during high speed running (44, 48, 145), all subjects ran at 20km/h, the maximum velocity of the available treadmill. It is accepted that this may not have been the subjects own personal maximum speed and this is acknowledged as a limitation.

4.3.5 Data processing

An overview of the data processing methods employed in this research is provided in the following sections. The matlab code related to each section is included in Appendix 9.2.1 for further information.

4.3.5.1 Kinematics

4.3.5.1.1 Marking strides

Following the inputting of participant anthropometric variables into the CODAmotion software, subject movement datafiles were converted to matfile format for further analysis. We wished to explore differences in movement patterns across the entire stride. Therefore, we set about dividing the entire 10 second record into strides by marking points of initial contact on the data. A custom Matlab (Version 2014b, Mathworks, Nantick, MA, USA) program was written for this purpose. The gold standard for marking strides in gait is based on force data usually from embedded force plates in the treadmill. Unfortunately, we did not have such equipment available to us and initial contacts were marked using kinematic data alone. Prior research has highlighted three approaches which can be used for this purpose during treadmill locomotion (146, 147). The most reliable method is to consider the horizontal velocity of the heel marker. The treadmill belt is moving backwards at a relatively steady speed. Therefore, the point at which the foot comes in contact with the belt can be determined as the beginning of a period of steady negative velocity on the heel marker velocity plot (146, 147). Furthermore, the vertical velocity of the heel marker is also influenced by the movement of the treadmill, and a period of steady zero velocity can be identified. Finally, the vertical position i.e. height of the heel marker, can also be used to identify the point of initial contact which equates to the lowest point of this graph. A graphic user interface (GUI) was created to enable the identification of initial contact and toe off using each of these methods (see Figure 16). The toe off data was not used in this

research but is described here for completeness. The points of initial contact were determined via visual inspection and marked by the assessor using the *ginput* function in the matlab graphics tool book. *Ginput* allows the user to identify points on a data record through a mouse click via the superposition of a cross hair over the GUI. The timepoints of all initial contacts for each record on both legs for each subject were identified and stored.

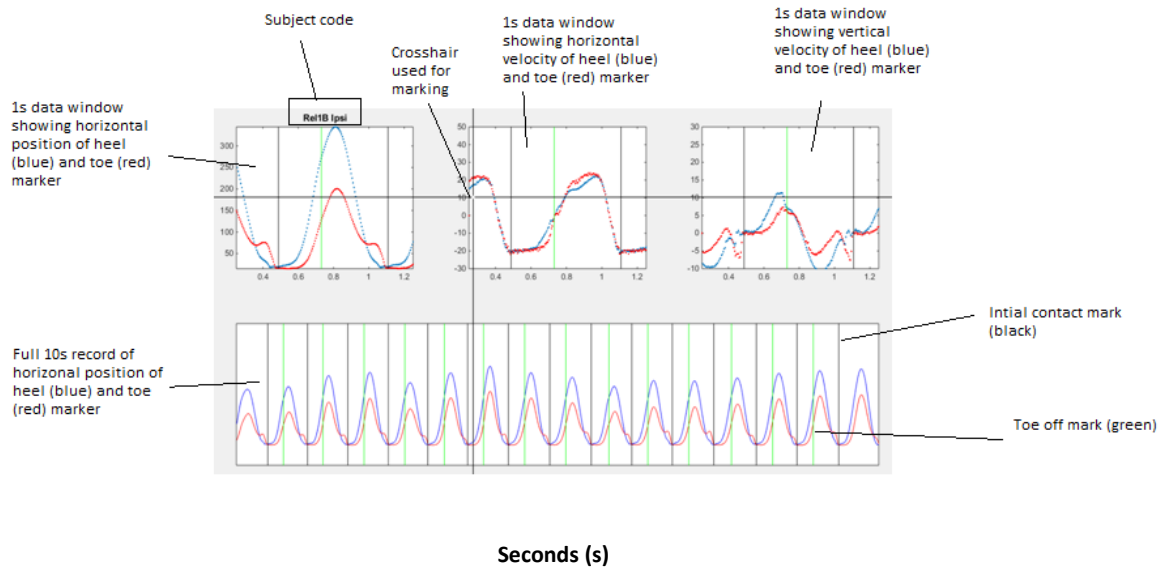


Figure 16: Matlab graphics user interface showing: height of the heel (blue) and toe (red) in the sagittal plane for one second of data (top left) and for the whole record (bottom); vertical velocity of the heel and toe marker in the sagittal plane for one second of data (top middle); horizontal velocity of the heel and toe marker in the sagittal plane for one second of data (top right). Initial contact marks are black vertical line and toe off marks are green vertical lines. The cross hair was used to place the marks using the *ginput* function in matlab.

4.3.5.1.2 Subdivision and averaging of kinematics

Using the points of initial contact, all kinematic data relating to complete strides were extracted and compiled for each limb in each subject (see Figure 17). Limbs were identified as either ipsilateral or contralateral, with ipsilateral equating to the previously injured limb or the side matched limb in controls. It was accepted that individual subjects would differ in their stride times and therefore in order to address this variance, stride kinematic data was normalised across 101 timepoints. This is a standard approach when assessing human gait (138). The mean stride kinematic data was calculated for each subject and saved. The symmetry between limbs was expressed as the ipsilateral data minus the contralateral limb data for each plane and joint. Mean segment rotation data for each limb and symmetry data for each subject (three planes and five joints) was collated according to group and

group means and standard errors calculated (see Figure 18). At all stages data were plotted in graphic form for visual inspection to ensure errors were identified in the calculation.

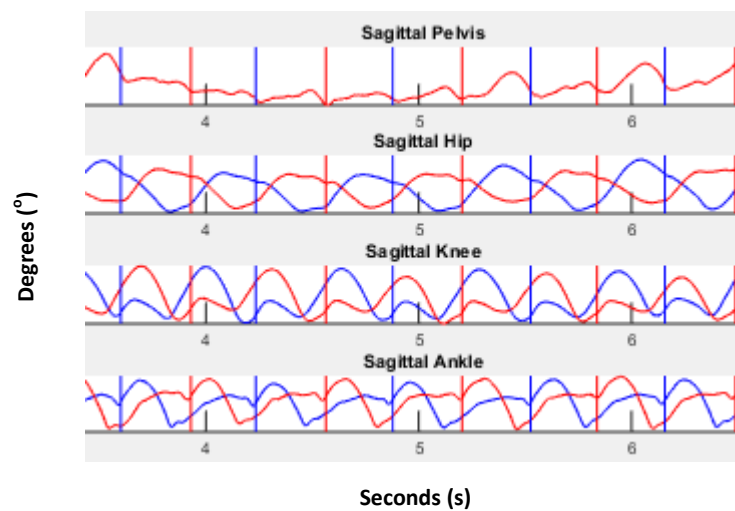
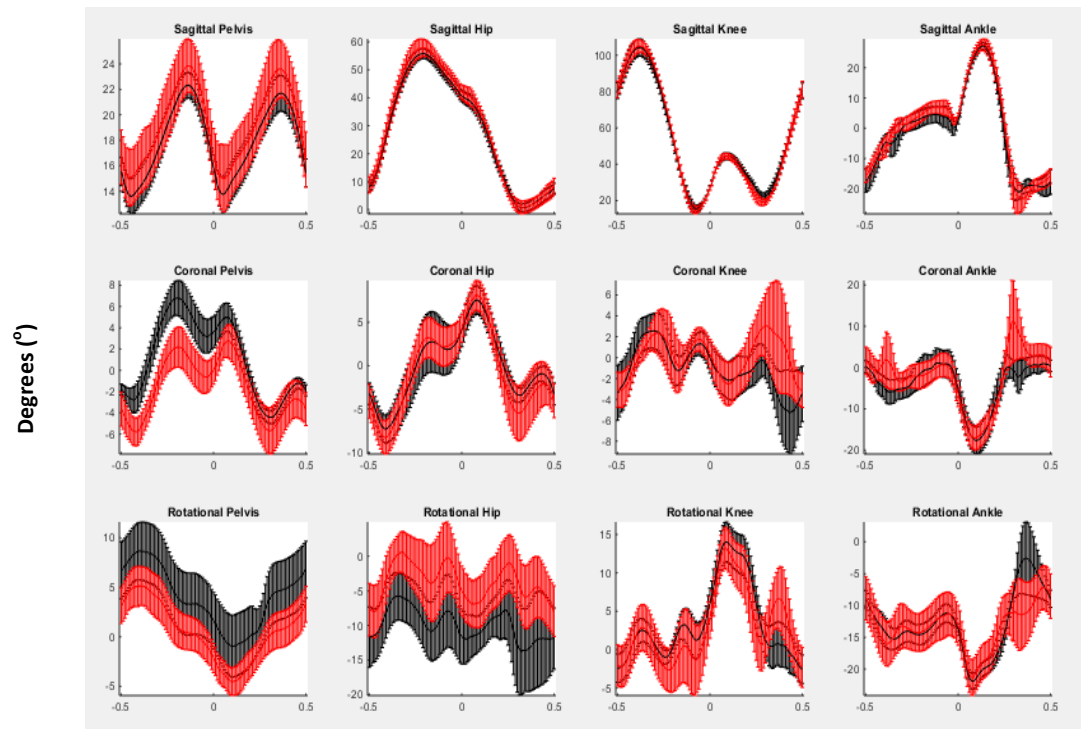


Figure 17: A selected subset of kinematic data for the sagittal pelvis, hip, knee and ankle rotation data. Movement traces are shown for left (blue) and right (red) limbs for one subject. Heel strikes are denoted by vertical lines (red for right, blue for left).

A decision was made to plot the time normalised data with initial contact placed centrally. This was considered appropriate as the period about heel strike having been identified as the point during the running gait cycle where the biceps femoris is most active and also most likely to injure (51, 72, 85, 148). Aligning the data in this way centred the visual focus on this time frame which otherwise would have been at the extreme left and right of the plots.



Stride across 101 normalised time points

Figure 18: Mean group kinematic data plotted for group one (red) and group two (black). Error bars = ± 1 standard error. X-axis 0 equates to initial contact.

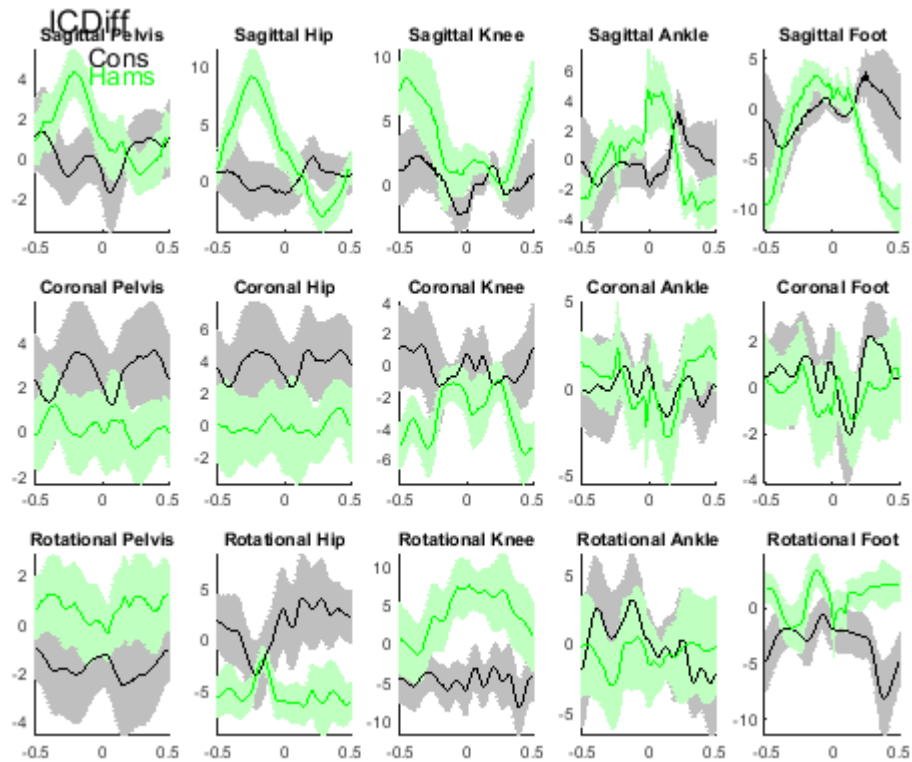


Figure 19: Symmetry data for all joints and planes. Group one (green) and group two (black). Error bars = ± 1 standard error. X=axis 0 equates to initial contact.

4.3.5.2 EMG

In order to remove any y-axis amplitude offset, the mean value of the signal was subtracted from the raw EMG data. This data was then full wave rectified by calculating the absolute signal values thus producing a signal which was comprised of positive values. The rectified signal was log transformed and subdivided according to stride using the same methods as the kinematic data (see 4.3.5.1.2 and Figure 20). This created an average and time normalised EMG signal across for each of the nine muscles collected. Furthermore, ratios were calculated for:

- Biceps femoris:ipsilateral gluteus maximus (BF:iGM)
- Biceps femoris:ipsilateral erector spinae (BF:iES)
- Biceps femoris:ipsilateral rectus femoris (BF:iRF)
- Biceps femoris:ipsilateral external oblique (BF:iEO)
- Biceps femoris:contralateral gluteus maximus (BF:cGM)
- Biceps femoris:contralateral erector spinae (BF:cES)

- Biceps femoris:contralateral rectus femoris (BF:cRF)
- Biceps femoris:contralateral external oblique (BF:cEO).

These muscles were selected as the main contributors to sagittal plane lumbo-pelvic-hip control which were suitable for surface EMG analysis. The data was collated according to previously injured and control groups and group means and standard errors calculated (see Figure 21).

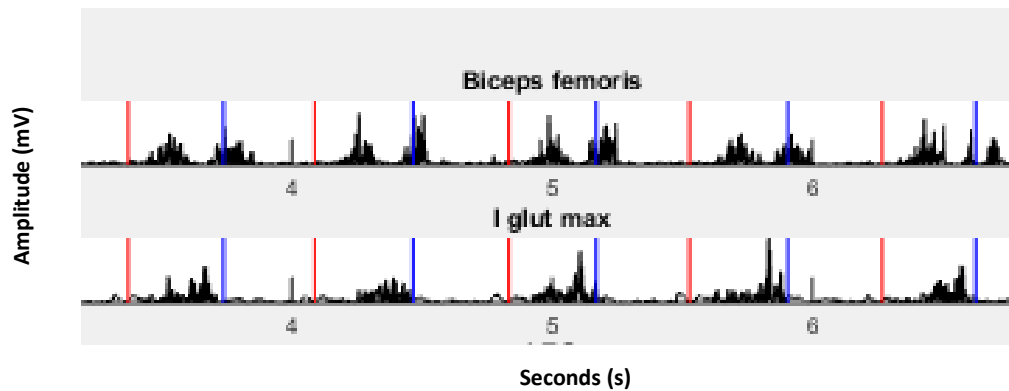


Figure 20: EMG record showing a selection of data for the biceps femoris and ipsilateral gluteus maximus (I glut max) with initial contacts marked using vertical lines for the ipsilateral (blue) and contralateral (red) limbs of a single subject.

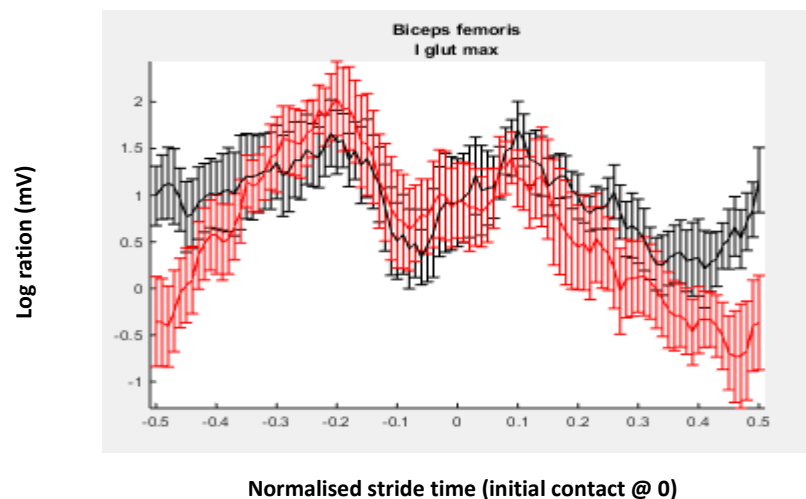


Figure 21: Representative example of the log EMG ratio of bicep femoris:ipsilateral gluteus maximus (I glut max) time normalised to running stride and plotted across 101 timepoints with initial contact at point 0 (centre). Red = group one, Black = group two. Error bars = +/- 1 Standard error of the mean for group.

4.3.5.2.1 Log transformation

A natural characteristic of EMG data is that the mean of the unrectified signal is equal to zero (130). Therefore, rectification of EMG signals results in a dataset which is skewed in

distribution. This has implications when exploring data using parametric statistical tests. We elected to log transform the data which served to realign the distribution and enable more robust testing with parametric approaches. This approach is supported by previously published data (149) and log transformation is a recognised method to compensate for non-parametric distribution of data.

4.3.5.2.2 The use of EMG ratios

Making intergroup and inter-individual comparisons using EMG data requires careful consideration of the naturally occurring differences in EMG signal characteristics occurring between individuals. The distribution of motor units in skeletal muscle is non uniform. Therefore, the EMG signal may be altered significantly as a result of the location of and density of fibres type between subjects. Also, the low pass filter effect of subcutaneous tissues may be exaggerated in an individual with increased body fat levels. For this reason, the EMG signal must be manipulated to account for this inter-participant variance, a process known as normalisation (150, 151).

The established approach to normalisation is to take a reference EMG amplitude from the participant and express test amplitudes as a percentage or ratio of the reference value. Typically reference values are calculated during maximal effort isometric contractions (MVC) or during the particular effort under consideration is chosen (e.g. mean or peak values) (150-152). However, these recommendations are in place for healthy muscle only. As the data in the projects relates to previously injured muscle, an alternative approach to data analysis was required. Specifically, one is unable to determine if a reference amplitude was a true indicator of muscle function, unimpeded by the effect of the previous injury.

The use of functionally relevant EMG ratios to explore for post-injury dysfunction was suggested by Edgerton *et al* previously (153). They suggest that through exploring the interaction between pairs of muscles which are involved in the same motor task, one can identify changes in recruitment patterns which may indicate compensations for underlying dysfunction. This approach has been used in the examination of muscular dysfunction associated with adductor related groin pain (154) and low back pain (155) previously. To the authors knowledge, no investigation of EMG ratio data has been conducted in subjects with previous hamstring injury *in vivo*. Using musculoskeletal modelling, Chumanov *et al*

(46) have highlighted the potential impact of alterations in muscle control about the pelvis on biceps femoris strain through late swing. In particular, they highlight the potential negative influence of contralateral iliopsoas and rectus femoris activation as well as activation of the ipsilateral erector spinae. Figure 22 is extracted from the work of Chumanov et al and indicates the influence of increased activation of various muscles on right side biceps femoris stretch. This highlights the synergistic nature of the various muscle that insert about the pelvis girdle, with particular focus on those influencing sagittal motion. We employed EMG ratio analysis to explore the synergistic relationship between sagittal plane influencing muscles with pelvic attachments that were assessable via a surface EMG approach.

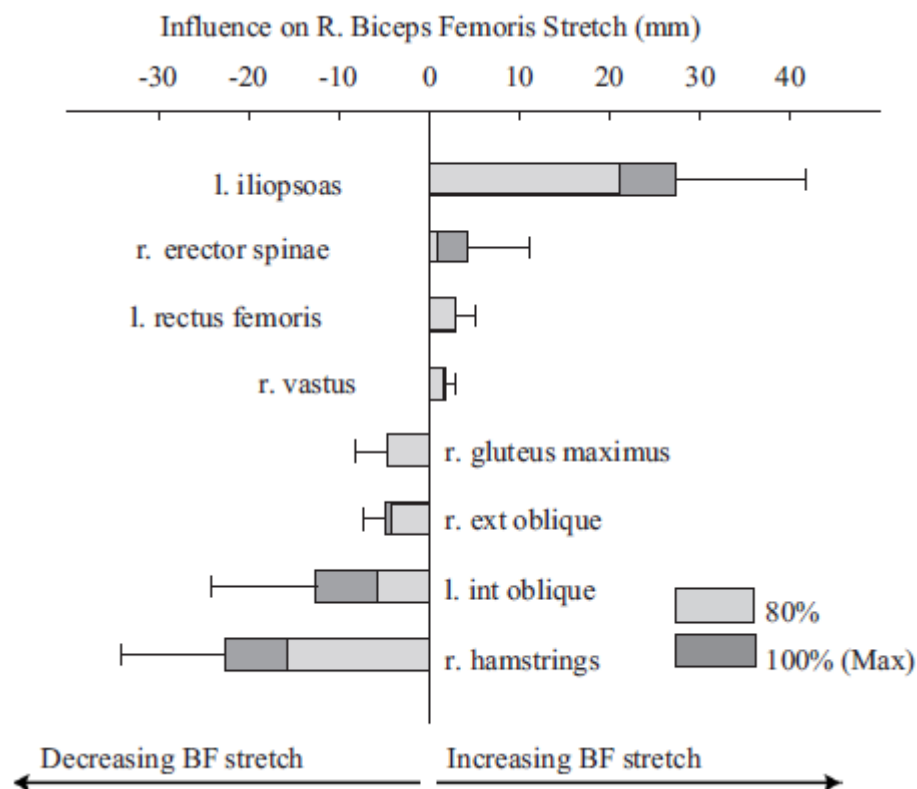


Figure 22: The muscles that had the greatest magnitude of influence on biceps femoris stretch during double float at 80% and 100% maximum effort sprinting. Taken from Chumanov et al (46)

We elected to make comparison between the previously injured biceps femoris and the other muscles which had greatest influence on sagittal pelvic position, and that were accessible via s-EMG analysis. For this reason, comparison was made with the gluteus maximus, erector spinae, rectus femoris and abdominal external oblique on both ipsilateral and contralateral limbs.

4.3.6 Statistical methods

Determining the most appropriate statistical tests to perform on this data was problematic due to the lack of methods in the literature, the novelty of the methodology and the wish to avoid point by point comparison in the dataset. In order to avoid the introduction of bias into the analysis, segmenting the stride data into sub sections or points for comparison was avoided. Maintenance of both the order and length of the 101 points making up the stride was also preferred. In the initial examination of the graphical representations of the data, it was noted that there were some apparent differences between previously injured and control groups in the late swing phase for some segment rotation and EMG ratios. Clear variations in the standard error plots between groups appeared to indicate significant difference. However, for the remainder of the stride, these differences were not apparent. Analysis of variance does not respect the time order of this dataset nor the fact that each timepoint is highly correlated with the preceding and following point. This approach would not be appropriate as significant difference across the entire stride was not anticipated. Similarly, autocorrelation would lack sensitivity for similar reasons. An alternative approach would have been to apply a series of student t tests at each timepoint. However, this approach would carry a high risk of Type 1 error, whereby a spurious finding would be considered as representative of group difference. This approach also requires multiple Bonferroni corrections to address this risk.

Instead, the analysis was focussed around the standard error of the difference between the groups. The standard error of the difference was calculated using the following formula,

$$SED = \sqrt{SE1^2 + SE2^2}$$

where SE1 equals the standard error of the mean of the control group and SE2 equals the standard error of the mean of the previously injured group data. This approach is advocated by Schwartz et al (138) when making comparative analysis of gait data. Using the SED, parts of the record where the difference in mean values for each group divided by the standard error of the difference exceeded 1 were identified. The area of any contiguous part of the record where such a magnitude of SED occurred was calculated, considering both the length in timepoints and amplitude of the difference. Finally, through a bootstrapping process, we made 1,000,000 comparative datasets by randomising the order of both the previously injured and control group across the 101 timepoints. We

determined how likely it was that a contiguous difference of the same magnitude as that seen in the true data would exist in the randomised datasets. This enabled us to calculate a p value which equated to the percentage of occurrences that such a difference emerged within the generated datasets.

Indeed, we found significant reductions EMG ratios in the BF:iGMax (maximum difference -12.5%, $p=0.03$), BF:iES (maximum difference -12.5% $p=0.01$), BF:iEO (maximum difference -23%, $p=0.01$) and BF:cRF (maximum difference -22%, $p=0.02$) in the previously injured group. These reductions were confined to the late swing phase. The kinematic data indicated that during the late swing, an increase in anterior tilt and hip flexion occurred on the injured side while reduced anterior tilt and hip flexion occurred on the uninjured side leading to maximum asymmetries of 4° ($p=0.02$) in pelvic and 8° ($p=0.01$) in hip movement. Transverse plane asymmetry of 6° (maximum) ($p=0.03$) was detected in the previously injured group in the knee joint during terminal swing and early stance phases.

4.4 Analysis of low density, high surface area EMG during eccentric loading

4.4.1 Selection of loading protocols

4.4.1.1 Case control study (Chapter 6).

We selected the Nordic exercise to examine the performance of athletes with previous hamstring injury. The Nordic is described as a mid to outer range eccentric focussed loading exercise specifically targeting the hamstrings. However, it is acknowledged that the exercise does not involve hip flexion and therefore the hamstrings are not lengthened to their maximal range. To perform the Nordic, the individual kneels in an upright position with the ankle secured by a training partner or restraint. They slowly lower their torso towards the ground without flexing at the lumbar spine or hips. In this way they eccentrically load the hamstrings beyond the point of failure. When they reach their maximal tolerable trunk inclination and their hamstrings can no longer resist, the torso falls towards the floor and the subject breaks the fall with their hands (see Figure 23). Petersen *et al* (64) carried out a randomised controlled trial where 942 professional footballers were allocated to groups with one exposed to a progressed regime of Nordics in addition to their standard pre-season and in-season training. They found that the rate of hamstring injuries per 100 player seasons was reduced by 71% in those who completed the Nordic programme. Van der Horst *et al* (156) report a 68.8% reduction in following season injury

rate in amateur footballers completing the Nordic protocol. This has resulted in the Nordic loading protocol being strongly supported as a preventative measure in hamstring injury in football (157) and has been incorporated into the FIFA 11+ injury prevention programme.

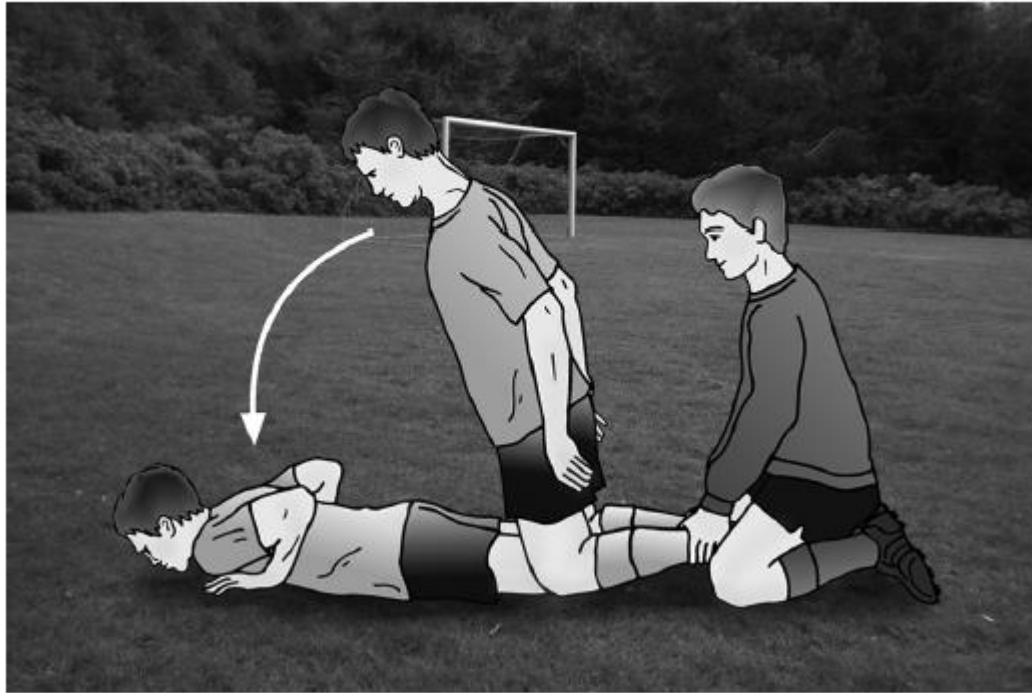


Figure 23: The Nordic exercise (taken from Petersen et al (64)).

4.4.1.2 Case studies (Chapter 7)

The selection of loading protocols for the subjects presented in case study form were based on minimising the risk of injury recurrence and providing information of immediate clinical benefit to the participant while also addressing the research question (Section 2.1.2).

Specifically, the aim was explore the acute and sub-acute stages of muscle rehabilitation in elite athletes using a selection of tools including low density EMG arrays, isokinetic dynamometry and other external measures of force. In both case studies, the participants were recruited from elite professional sport. They were also both approximately five weeks post injury. We were therefore guided by their treating clinician as to the suitability of various test protocols at our disposal. In the case one, we elected to assess force and EMG activity during isokinetic testing. There is a reasonably large body of research which has examined isokinetic profiles in previously injured subjects. This information is summarised

in Chapter 3 and indicates persistent reduction in torque during slow eccentric knee flexor contractions. This information provided a comparative baseline to extract some short-term clinically relevant information about the participant's performance in comparison to known norms. The participant was tested using contraction velocities based on the work of Croisier et al (32) who conducted one of the only prospective analyses of isokinetic associated hamstring injury risk. Specifically the athlete performed a 60 and 240°/second concentric and 30 and 120°/second eccentric knee flexor contraction to match their methods.

With the second case, the athletes medical team were cautious regarding exposure to less controlled maximal load. This participant had sustained a repeat hamstring injury shortly after a preceding injury and any risk of recurrence was to be avoided. For this reason, force and EMG activity was measured during isometric contractions. The participant had full control and could modulate his effort to ensure a comfortable contraction if necessary. The hamstring was tested at three functional lengths – the knee in an extended position, the knee flexed to 45° and the knee flexed to 90° in order to explore the characteristics within each range. The hip was held in neutral insofar as possible while the subject lay prone.

4.4.2 Measuring muscle activity using EMG sensor arrays

4.4.2.1 Background

Measuring EMG using bipolar methods is inherently limited by the relatively small detectable range under the electrodes (130, 158). At best the resultant signal can be described as an approximation of muscle activity in the muscle or muscles underlying the sensors. However, muscles are known to display non-uniform EMG activity in response to loading. The distribution of muscle fibre types, the architecture of the underlying muscle fibres, the location of the motor end points, the distribution of motor units and the nature of the demands being placed upon the muscle, will all have a bearing on the characteristics of myoelectric signal in the area of the sensor (158-162). Several studies have explored the regional distribution of EMG activity using low density, high surface area arrays of sensors with outcomes related to fatigue and delayed-onset muscle soreness predominating (159-163). None, to our knowledge, have explored spatial activation patterns in previously acutely injured muscle.

4.4.2.2 Development

The aim of this study was to explore spatial activation patterns in previously injured hamstring muscles. A 64 channel EMG system (REFA, TMSI, Netherlands) was employed. This system was available in the Queen Mary University of London human performance laboratory (Figure 24). This system also has the benefit of a number of auxiliary inputs which allowed for the simultaneous collection of force and other signals.

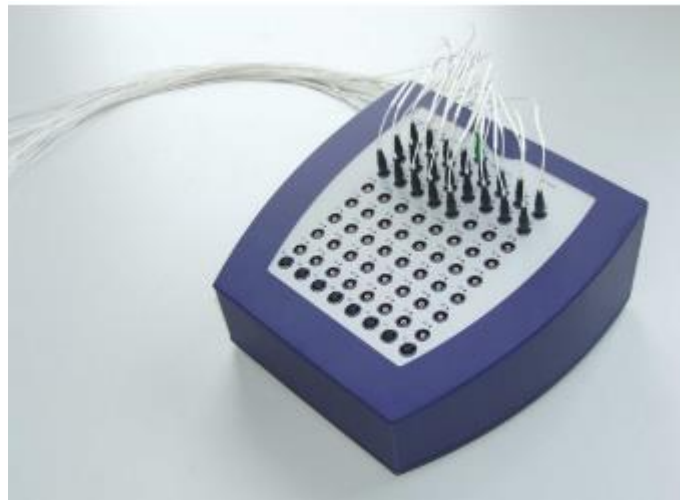


Figure 24: The REFA 64 channel system (TMSI Netherlands)

A linear multichannel EMG arrays was created using the micro-electrodes which are available with the REFA system (Figure 25). These sensors have a 1mm silver silver chloride sensing area. The small sensing area is suggested to minimise crosstalk and motion artefact. Through a series of experiments, a method was developed to fix these sensors to the skin. Although each sensor could be positioned independently, we wished to standardised our approach. Therefore, linear arrays, four sensors long, were created using a combination of hypoallergenic tape (Hypafix, BSN Medical) which was in contact with the participant, backed with double sided adhesive tape which was in contact with the electrode. The strips of tape were perforated at 20mm intervals using a 5mm punch. A syringe was then used to apply a small amount of conductive gel to the surface of the sensor. Finally, the arrays were further secured to the skin with surgical tape (Meditrans, Fleming Medical Ltd.). This method was developed by the primary researcher alongside engineering colleagues.

The 20mm inter-electrode distance was chosen in order to maximise the spread of the sensors over the area of the hamstrings while maintaining a standardised approach. This distance would also reduce the influence of crosstalk from the region under the adjacent electrode.

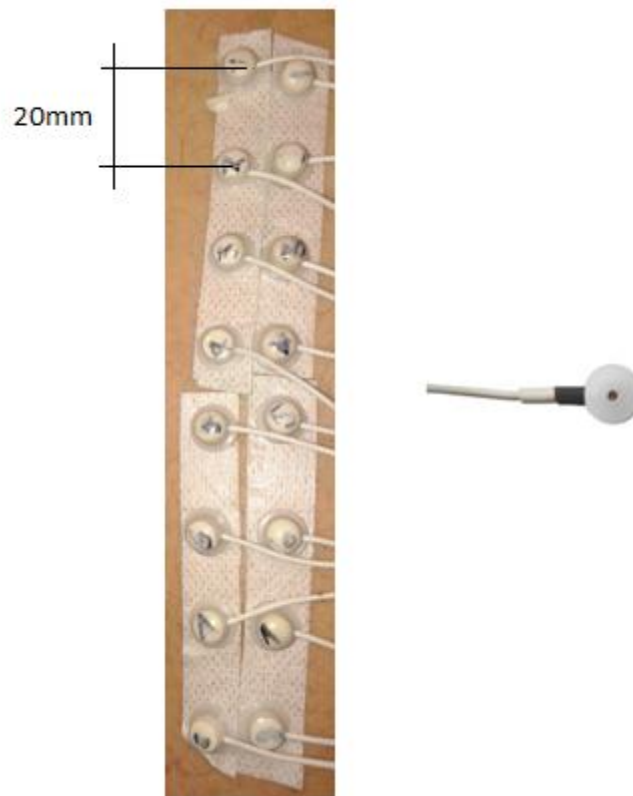


Figure 25: A micro-electrode from the REFA system (right) and two linear arrays in situ on a subject.

4.4.2.3 Implementation

In order to collect data from functionally relevant areas, the electrode placement guidelines from SENIAM for bipolar s-EMG positioning were consulted (136). The midpoint of the biceps femoris was identified as a point 50% along a line drawn from the ischial tuberosity to the lateral epicondyle of the tibia. The midpoint of the combined medial hamstring (semitendinosus and semimebranosus) was identified as a point 50% along a line drawn from the ischial tuberosity to the medial epicondyle of the tibia. These two points were used to orientate the positioning of the linear arrays. On the skin overlying the biceps femoris, four 4 channel linear arrays were attached with two lying proximal to the muscle

midpoint and either side of the muscle line. Two further arrays lay in a similar arrangement distal to the muscle midpoint taking care to ensure the electrodes either side of the muscle midpoint has a 20mm spacing. Thus, the total composition of each linear array was 8 electrodes with 4 arrays on each leg, giving a total of 32 sensors per hamstring. The arrays overlying the medial hamstrings followed a similar arrangement. Channel numbers matched the specific location over the hamstrings in each leg/subject. A schematic representation of electrode placement is provided in Figure 26. We also ensured accuracy of placement by palpating the muscle edges and testing the activation in real time during a resisted knee flexion effort as per published guidelines (130, 131, 164).

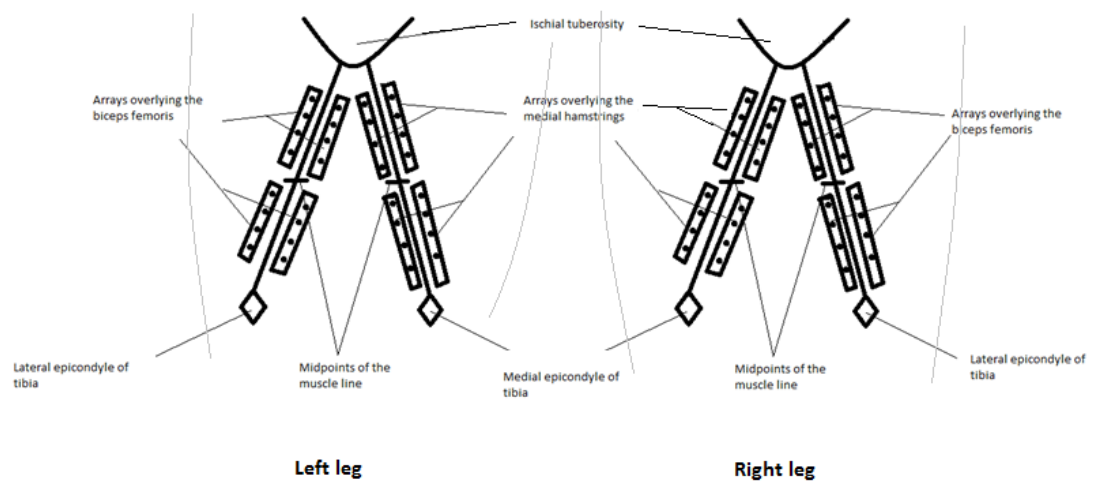


Figure 26: Schematic representation of linear electrode array positioning over the right hamstrings viewed from a posterior direction.

4.4.3 Force measurement during the Nordic

4.4.3.1 Background

There is a growing body of research which has explored instrumented measures of force during the Nordic. This research is almost exclusively produced by a group led by Dr Anthony Shield in Brisbane. This group has developed a device called the NordBord (see Figure 27) to measure strain via two independent strain gauges which are attached to braces which stabilise the ankles in subjects as they perform the Nordic. The NordBord has been commercially developed and marketed to sporting organisations around the world. This device has been shown to be reliable in measuring strain about the ankles during the Nordic (121). The device has also been used in detecting force asymmetries and reduced

pre-season strength gains in athletes with previous hamstring injury (65, 68) . We sought to replicate this device for our experiments with the added measure of low density, high surface area EMG to more thoroughly investigate athlete muscular performance during the Nordic.



Figure 27: The NordBord promotional material.

4.4.3.2 Development and implementation

In collaboration with the School of Engineering and Material Science, Queen Mary University of London, a version of the NordBord was fabricated for use in these experiments. Two independent s-type strain gauges (TSA Alloy Steel, Coventry Scales Company, UK) were attached to steel d-rings. Several d-rings were screwed to a 20mm ply board which provided a rigid anchor for the strain gauge. To ensure flexibility in attachment position, d-rings were screwed into the ply board at various widths. The strain gauges were fitted with an eyelet. We clipped a small climbing caribina to this eyelet, which in turn was attached to the d-ring (see Figure 28). We used a similar arrangement of eyelets and caribinas on the subject end of the strain gauge. Instead of attaching this end

to d-rings, we clipped on a heavy duty ankle cuff used for gym based weight training. This was adjustable and was strapped to the subject's ankles (see Figure 29).

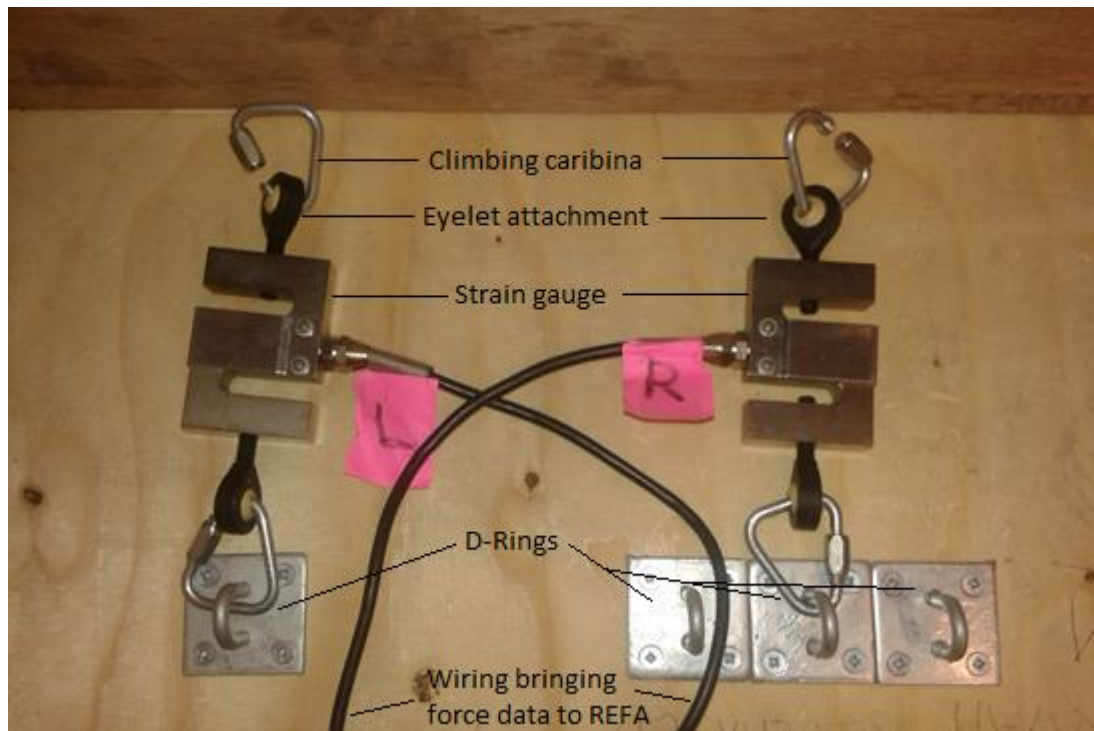


Figure 28: Picture of setup associated with strain gauges in the Nordic force measurement device

The signals collected from both the left and right strain gauges were collected by the REFA systems via two auxiliary ports via a 5 pin binder connector. We conducted separate tests of the signal outputs from each strain gauge. A known mass was attached to the gauge and the signal amplitude calculated. This was completed for three separate masses and a conversion factor to kilogram weight was devised. The signal also required re-integration and experiments where the known force was rapidly returned to zero were conducted to measure the integration factor.

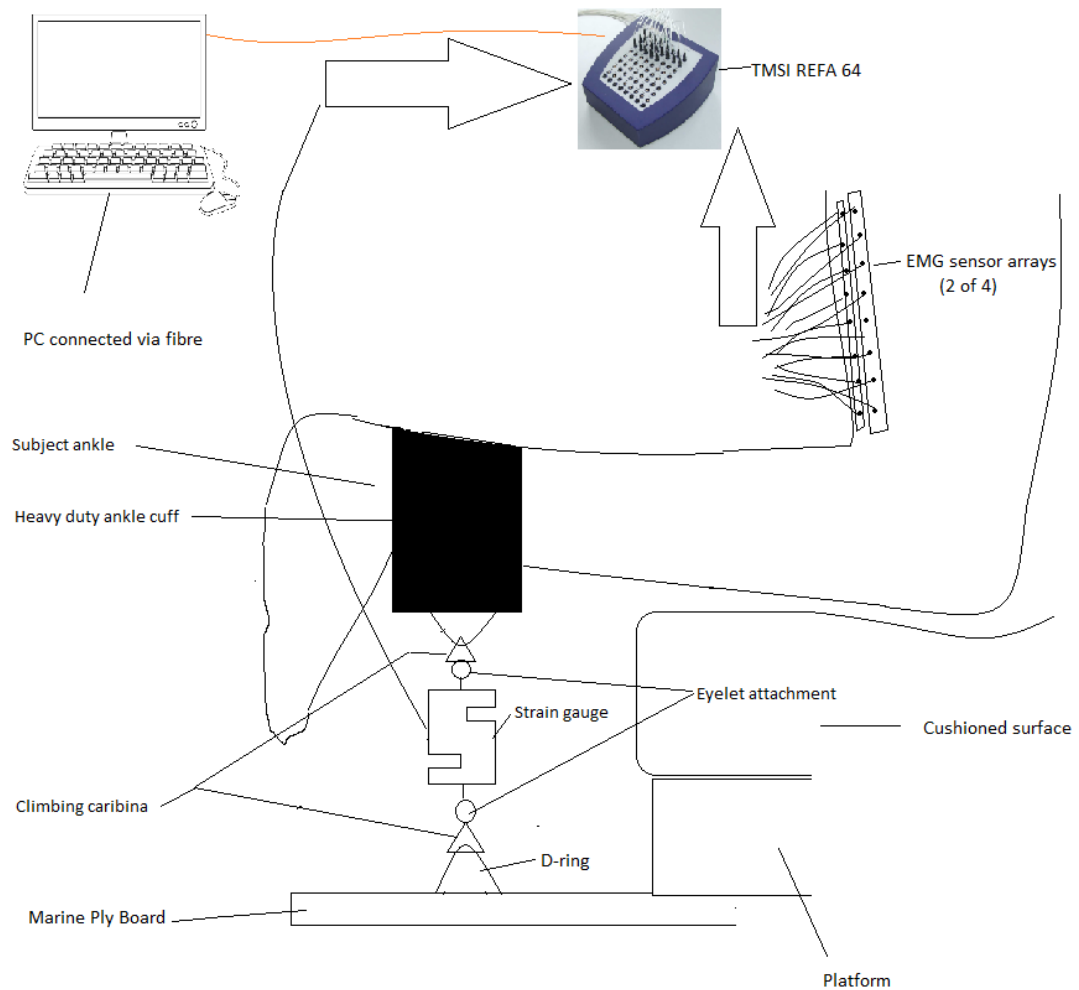


Figure 29: Arrangement of the Nordic data collection set-up

4.4.4 Data acquisition

All low density, high surface area EMG signals and force signals were collected at 2048hz using PortLab software (TMSI, Netherlands) on a separate PC via a two-way glass fibre link. Data was then converted to matlab format for off line analysis. Within the REFA system unipolar signals are amplified against the mean of all connected channels. All EMG cables were individually shielded to ensure minimum interference from ambient signals or motion artefact. We live monitored all signals during data collection and made adjustments if we detected any signal interference due to poor contact with the skin. The ground electrode was attached to the subjects calcaneum.

A standardised approach was used to try and maximise the repeatability of the force measurements. The subject knelt on a cushioned platform with their malleoli positioned directly above the row of d-rings on the plywood base. The strain gauge was orientated

perpendicular to the plane of the tibia and clipped in place with the caribinas and ankle cuffs. Any adjustments were made to ensure maximal comfort for the participant. Signal quality was checked in advance and during data acquisition.

4.4.5 Data processing

The matlab code related to each section is included in Appendix 9.2.2 for further information.

4.4.5.1 Force data

The force signals were collected in integrated form. Following reintegration, signals were converted to mass using the conversion factor from the aforementioned experiments (Section 4.4.3.2). Force data was plotted and peak force was calculated for each limb in each subject.

The force records were also used to determine the onset and offset times of the Nordic exercise for some of the EMG analysis. The onset of the Nordic exercise was found to be characterised by a steady rise in force with a clear onset time. The incremental increase in force continued to the point of max force after which there was a high velocity reduction in force as the subject fell forwards. Both the change in pitch (signifying velocity) and direction of the force slope were clearly identifiable on the record. The beginning and end of this period were marked using the matlab *ginput* function which enables the user to indicate points along the x axis of a graphic with a mouse click. In many cases, following the rapid reduction in force as the subject fell forwards, there was a momentary increase in force as the subject broke their fall assisted by the hamstring. The end of the exercise was marked before the increase (see Figure 30).

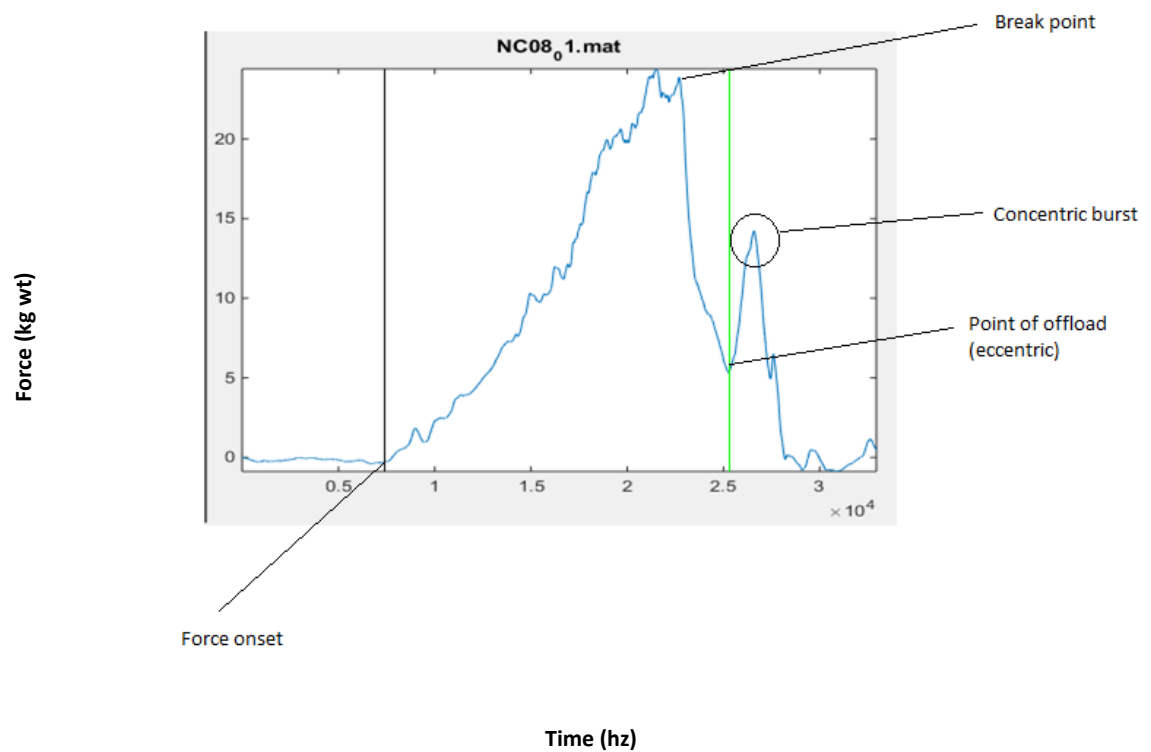


Figure 30: Representative example of a force record during the Nordic. The black vertical line is positioned at the onset and the green vertical line at the end of the loading period.

4.4.5.2 EMG data

EMG signals were band pass filtered at 10-500hz in order to remove frequencies unrelated to muscle activity as per international guidelines (136, 164). Furthermore, a notch filter about 50hz was applied to ensure removal of ambient electrical interference. Individual signals were examined for quality through visual inspection of the filtered data (see Figure 31). Data was removed from further analysis when motion artefact adversely affected signal quality (low frequency, high amplitude signal) or where data was missing due to detached/damaged sensors. Finally, the signals were full wave rectified.

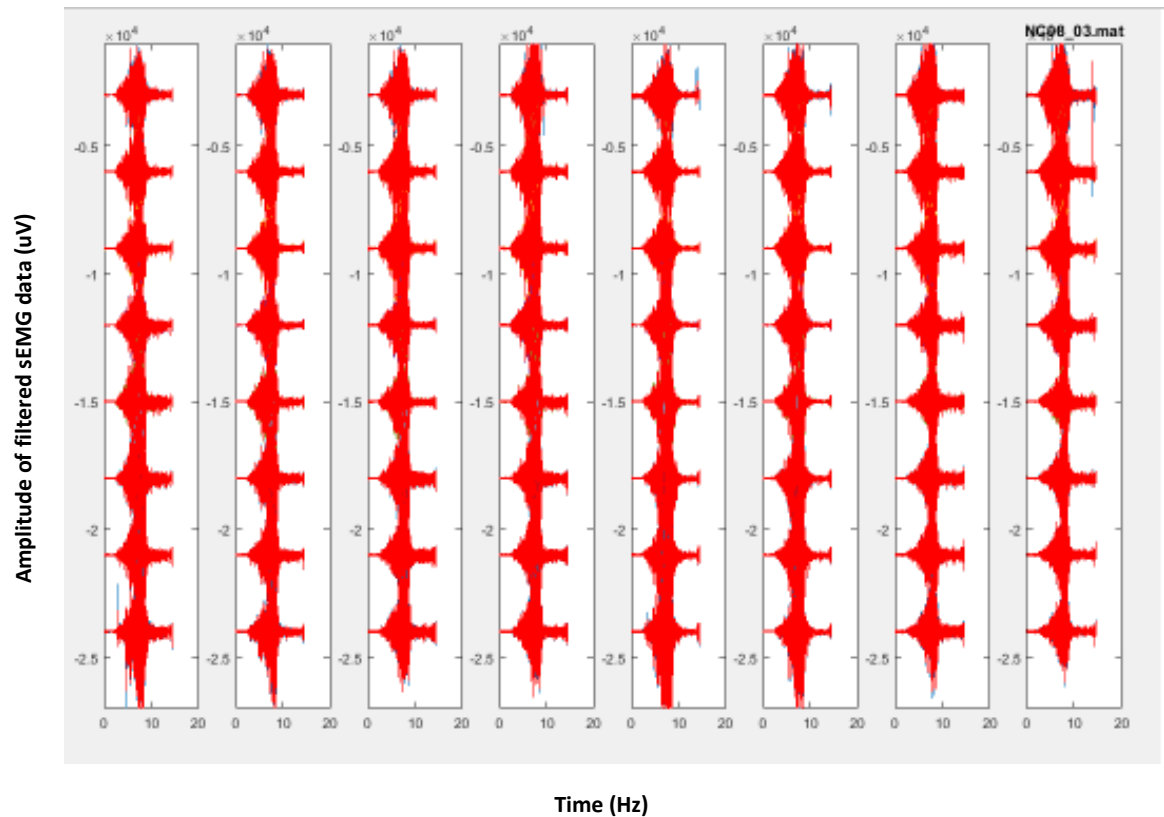


Figure 31: Representative example of filtered EMG data plotted in red and aligned according to the anatomical location of the sensor. Columns 1-4 = left side (lateral to medial). Columns 5-8 = right side (medial to lateral). This graphic was used to visual inspect for quality/

Extrapolation of clinically meaningful EMG data was performed using four separate methods outlined below. Inter-group comparisons were made between previously injured and uninjured limbs of previously injured subjects. In addition, intra-group comparisons were made between the previously injured and uninjured limbs of the previously injured group and the dominant limbs of the control group. Dominance was defined according to preferred kicking limb (165, 166).

4.4.5.3 Normalised EMG signal at time of maximal force

The filtered EMG data was rectified and smoothed using the Root-Mean-Square (RMS) method with a non-overlapping running 50hz window applied for the calculation. Smoothing of EMG data allows for clearer interpretation of findings and reduced the risk that momentary high amplitude signals will skew the data leading to interpretation error. In order to preserve the characteristics of the original EMG single in so far as possible, a 50hz smoothing window was chosen as this is the minimum recommended window size for RMS calculations (130). The RMS EMG signal was found to lack a clear onset and finish

time owing to the subjects performing isometric contractions at the beginning and end of the Nordic loading exercise. Using the force onset and finish times, the RMS EMG data corresponding to this period of loading associated with the performance of the Nordic loading exercise was extracted for analysis.

As described in 4.3.5.2.2 normalisation of EMG signals in previously injured may be an unsuitable approach. However, in order to present a thorough examination of EMG data and to conform the well-established principles, the RMS signals for each sensor were divided mean RMS during the loading period. Peak normalised RMS amplitude was then calculated for each channel. This approach conforms to standard methods for surface EMG signal processing (130, 152).

4.4.5.4 Channel by channel contribution to maximal EMG activity

The aim of the second analysis was to explore the contribution of each channel to overall maximal hamstring EMG signal and thus provide a spatial overview of hamstring muscle activity during the 'Nordic'. A novel procedure was developed to achieve this as follows. The mean activity across all 32 channels on each leg was calculated and smoothed by interpolating within a non-overlapping 25ms window. The period of maximal activity across all channels was defined as all timepoints within the top 20% of the total and smoothed mean. The activity for each channel occurring during this timeframe was extracted. Finally, the filtered and rectified amplitude data for each channel was expressed as a ratio of the total activity occurring across all channels, thus providing a ratio of activity at each sensor (Figure 32).

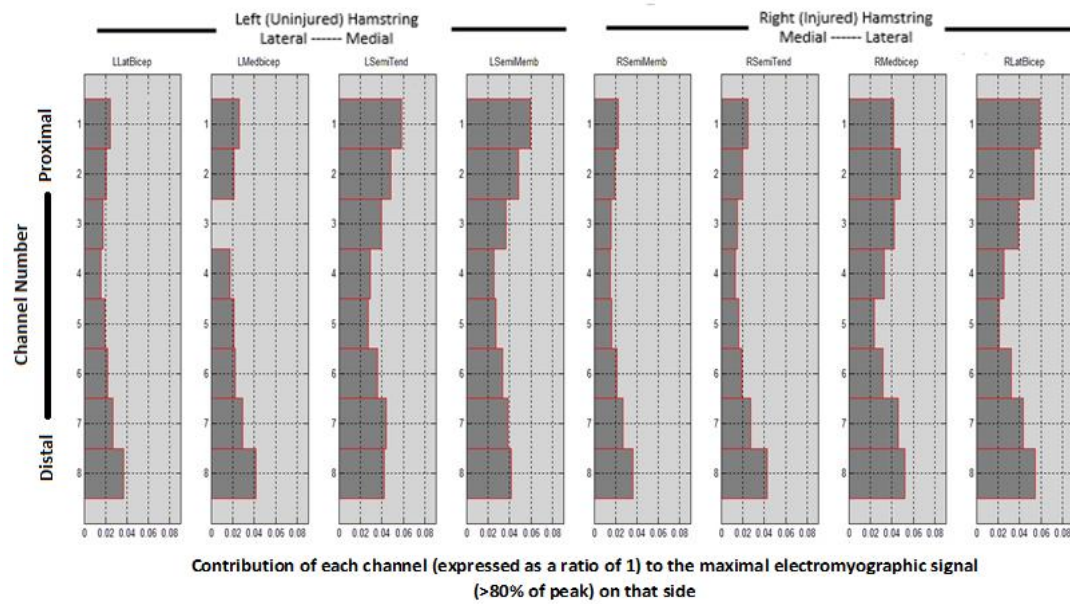


Figure 32: Ratio of contribution from each channel location to maximal EMG activity on the left and right limbs respectively

4.4.5.5 Integrated EMG activity (iEMG)

As the duration of loading varied between participants, the RMS EMG data was time normalised by interpolating all data to the number of samples in the longest duration sample. This enabled calculation of iEMG values without confounding influence from contraction duration. iEMG calculations were performed on the time normalised RMS EMG data using the trapezoidal method to calculate the area under the curve of the signal for each channel. The area under the curve of the RMS signal provides a representation of the overall volume of the muscle activity (130). The decision to normalise the RMS signal for time is somewhat unconventional but we could not control the duration of the Nordic and therefore felt is necessary to normalise according to contraction duration. Resampling to the longest duration signal eliminated the risk of data loss using the alternative of shortening.

4.4.5.6 Frequency Analysis

The median frequency for the EMG signal of each channel was calculated. The frequency spectrum provides information about the underlying nature of the motor units contributing to overall signal with larger fast twitch unit thought to fire at higher frequencies. The filtered, unrectified signal was extracted for the duration of the Nordic loading exercise, using the force onset and offload times as described above. A discrete Fourier transform

was performed across the entire signal (i.e. at 2048hz) to extract the frequency components. The median frequency was subsequently calculated for each channel.

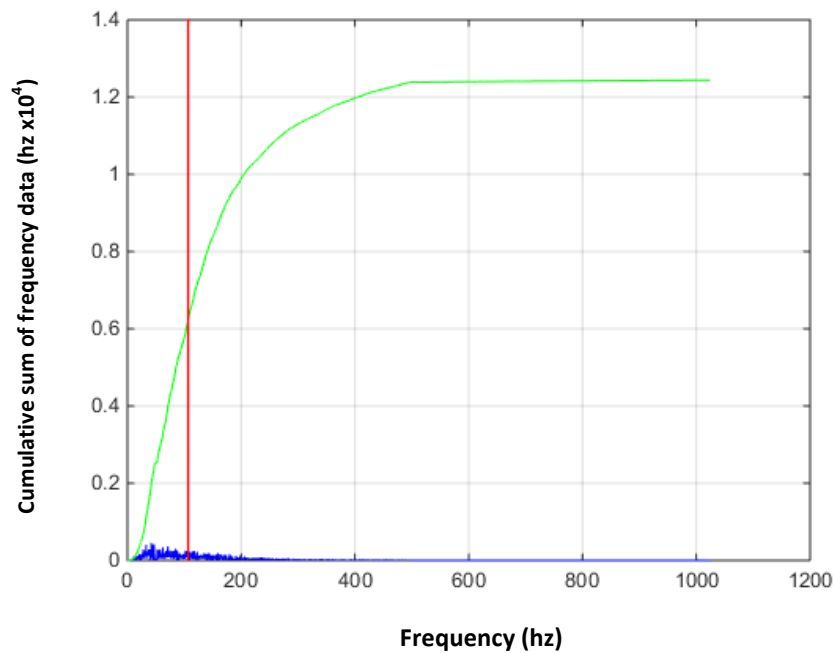


Figure 33: Frequency calculations for one channel of EMG data. Blue = power spectrum. Green = cumulative sum of power spectrum, red= median frequency.

4.4.6 Statistical methods

The distribution of the data for the force and each of the EMG outcomes was examined using a Shapiro-Wilk test as well as plotting the histogram of the distribution. We expected the EMG data to be skewed in nature and therefore were cautious about employing parametric approaches to statistical analysis. However, non-parametric tests do not facilitate the testing of factorial interactions. Previous research using low density s-EMG arrays has made comparison between study groups using three-way analysis of variance (163). The spatial location of the electrode was considered by inputting the horizontal and vertical location of the sensor as factors. In order to avoid type 2 error, we also performed two-way ANOVAs considering the horizontal and vertical location separately. This approach was used on both the original dataset and on a dataset which was rank ordered based on the factorial interaction(s) under consideration. This approach is known as aligned rank transform and is described in previously published statistical research (167-169). Indeed Wobbrock et al (167) provide a downloadable tool which performs these complex calculations on Comma Separated Value data. We used this approach to confirm

the findings based on the ordinal dataset and ensure we were not risking Type 1 error secondary to skewed data.

5 Observational Study: Running

The biomechanics of running in athletes with previous hamstring injury: a case control study

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5.1 Introduction

Hamstring muscle injuries are common in sports which involve sprinting (23, 60) with recurrence rates across various sports ranging from 12-41% (1, 8, 170, 171). Hamstring muscle strain is the most prevalent elite level injury in sports such as soccer, rugby, 'Australian Rules' football, Gaelic football and hurling (2-5, 8). The long head of the biceps femoris is the most commonly injured component (1, 172), with the late phase of swing in high speed running being a key risk phase of gait (46, 60, 85, 148). During this phase, the muscle is rapidly increasing in length while performing a high level of eccentric work in order to decelerate the shank for foot strike (85). This dual role is thought to be responsible, at least in part, for high vulnerability to injury.

Previous injury is the main statistical predictor of re-injury (173), for reasons which are incompletely explained. Emerging theories have suggested persistent post-injury neural inhibition as a mechanistic factor (61). There is a growing evidence base supporting this construct, with selective eccentric weakness and atrophy highlighted as possible manifestations of post hamstring injury neuro-inhibition (61). Determining what, if any, biomechanical factors are associated with prior hamstring injury during running and developing a method of identifying such factors may enhance management, screening and injury prevention strategies.

The aim of this project was to examine the biomechanical features of sprinting in athletes who had returned to sport following non-contact, sprint related hamstring injury in order to inform rehabilitation and prevention strategies. The objective was to recruit two groups of athletes; one with prior hamstring injury and their closely matched uninjured teammates, and make inter-group comparisons of activity levels in the biceps femoris with respect to functionally associated muscle groups, and lower limb kinematic symmetry, across the full stride during sprinting. The null hypothesis was that there would be no

difference between groups with respect to muscle activation ratios or kinematic variables across the stride. The alternative hypotheses were that there would be persistent evidence of hamstring inhibition and that intra-subject kinematic asymmetry would reflect this inhibition.

5.2 Methods

5.2.1 *Participants*

Ethical approval for this study was obtained from the University College Dublin research ethics committee, and all participants provided written informed consent prior to inclusion. Nine elite level male hurlers with a history of acute sprint related hamstring injury within the previous two years, and eight uninjured male controls volunteered to take part. Hurling is a rapid, grass based sport played at the elite level in Ireland (4). Each participant in the previously injured group was paired with a teammate of similar age, height and playing position. Control participant selection was therefore closely matched to account for variations in training demands and exposure. We were unable to match one previously injured athlete to a control subject.

All previously injured participants fulfilled criteria consistent with a retrospective diagnosis of an acute non-contact hamstring muscle injury within the previous 24 months (48, 127). Specifically, previously injured participants reported: (a) sudden onset of posterior thigh pain which occurred without contact and during high speed running, (b) pain which was localised to the hamstring region, (c) active hamstring contraction and running causing discomfort for a minimum of 48 hours following injury, (d) an inability to participate in sport for at least 48 hours following injury. As a secondary means of injury confirmation, participants were questioned regarding the presence of bruising following injury, whether they underwent contemporaneous assessment by a registered physiotherapist/medical practitioner and/or confirmatory imaging. The time since injury was recorded.

Participants were excluded if they had failed to return to full pain-free participation in sporting activity or had residual symptoms of hamstring muscle functional impairment (deficits during manual muscle strength testing (126)), sacro-iliac joint or lumbar spine dysfunction (positive sacral distraction and sacral thrust test (174), reduced or painful range of motion or discomfort during lumbar inter-vertebral movement testing (126), or

impaired neural dynamics (127). Participants in both groups were excluded if they had positive findings on completing the Physical Activity Readiness Questionnaire (175), had a history of lower limb surgery in the two years prior to recruitment or were not currently elite competitors. Control participants were excluded if they had a lifetime history of hamstring injury. Given the retrospective nature of this study it was not possible to match participants for pre-injury maximal running speed. Instrumented measures of strength were not included as part of the study protocol as this would have involved a separate visit to the laboratory as this outcome was not central to the stated study aims. Nevertheless, the omission of an instrumented measure of strength is acknowledged as a study limitation.

5.2.2 Experimental Protocol

Participants were required to attend on one occasion for testing. All testing took place in a university movement analysis laboratory. Participants were all familiar with treadmill running and ran at 20km/h without incline, in their own shoes, after a warm-up consisting of an incremental increase in running speed over five minutes until the 20km/h speed was reached. When steady state running was achieved (after ~ 1 min), data was collected for a 10 second period. The subject lifted themselves off the treadmill belt immediately on completion of the data capture period following verbal instruction from the assessor.

5.2.2.1 Kinematic measures

Participant movement data was collected for 10 seconds of steady running at 200Hz using an active infrared motion analysis system (Codamotion, Charnwood Dynamics LTD., UK) with markers placed on standard pelvic and lower limb anatomical landmarks using the modified Helen-Hayes protocol (128). Markers were firmly attached to the skin using double sided adhesive tape reinforced with transparent surgical tape.

5.2.2.2 EMG measures

Prior to placement, the skin was marked, shaved, abraded and cleaned with an alcohol wipe and electrodes positioned as per SENIAM guidelines (136). Pre-amplified silver-silver chloride electrodes with a 20mm inter-electrode distance (Motion Lab Systems Inc. LA. USA) were attached to the skin over the biceps femoris muscle of the previously injured hamstring, bilateral gluteus maximus, rectus femoris, abdominal external oblique and lumbar erector spinae muscle bellies. Specifically, the electrode for the biceps femoris was

placed centrally on the long head muscle belly at the midpoint between muscle origin and insertion. The anatomical borders of the muscle were confirmed by palpation and the EMG signal checked during contraction to ensure accuracy of placement. A similar procedure was adopted for all other muscle placements. Wiring was secured using a cohesive wrap, applied circumferentially around the chest, but ensuring that the participant did not feel impeded by the wiring or the wrap. EMG and motion data capture were synchronised using a digital switch (HLT100 INISO, BIOPAC Systems LTD., CA. USA). The EMG data were collected at 1200Hz for 10 seconds during the 20km/h trial.

5.2.3 Data analysis

Data was analysed offline using a custom written Matlab program (Version 2012, Mathworks, MA., USA). Foot strike was identified using the horizontal (Y-Plane) heel marker velocity, specifically the point when the heel began to move backward at a steady velocity on the treadmill, in agreement with previously described methods (147). Heel strike was further confirmed by cross-checking this mark with the point at which the heel ceased its descent onto the treadmill on the vertical velocity plot (Z-Plane) (147). EMG and kinematic data were aligned to heel strike, were interpolated and plotted across 101 time points for each stride in agreement with previous methods (154, 155). Comparisons were made between previously injured (ipsilateral) and uninjured (contralateral) limbs. Control participant limbs were designated as ipsilateral or contralateral based on participant matching.

EMG data was bandpass filtered at 10-500Hz and inspected for quality, rectified and logarithmically transformed. As is commonly found with EMG data, the distribution was strongly skewed towards negative values but conformed closely to a log distribution hence the decision to transform prior to statistical analysis. Any EMG data adversely affected by movement artefact or other external interference were rejected. Mean values and standard errors of the mean were calculated across all strides for each muscle. The mean and standard error of the following ratios were calculated from the processed data for each participant for all stride time points, averaged and collated for each group and presented as the following functionally relevant ratios: biceps femoris:ipsilateral gluteus maximus (BiFem:ipsiGluMax), biceps femoris:ipsilateral lumbar erector spinae (BiFem:ipsiErecSpin), biceps femoris:ipsilateral abdominal external oblique (BiFem:ipsiExtObl), biceps

femoris:contralateral gluteus maximus (BiFem:contraGlutMax), biceps
femoris:contralateral lumbar erector spinae (BiFem:contraErecSpin), biceps
femoris:contralateral abdominal external oblique (BiFem:contraExtObl). The use of EMG ratios, already normalised to gait phase, for comparative purposes accounts for inter-participant variability in signal amplitude and is in line with previously published data (154, 155, 176).

Kinematic data was segmented for stride using the foot contact data. Three dimensional kinematic measures (sagittal, coronal and transverse planes) for the pelvis, hip, knee and ankle were calculated (CODAMotion software v.6.76) and combined to provide group mean and standard error values. Movement symmetry was calculated by subtracting the averaged contralateral (non-injured) group stride values from those of the ipsilateral (injured) side for all joints and planes.

5.2.4 Statistical analysis

Anthropometric data, age and training exposure data were checked to insure normality of distribution and compared between groups using unpaired Student's T-Tests. Statistical tests were performed using SPSS software (Version 22, SPSS Inc. IL. USA) with significance set at $p < 0.05$. The mean and standard error of the group EMG ratios and kinematics were plotted using Matlab. As this was time series data, consecutive points are highly correlated and therefore unsuited to statistical testing using analysis of variance. We instead employed a bootstrapping technique to explore the probability of revealing, by chance, a contiguous length of record with a difference between groups as large (same or greater peak difference and same or greater area of difference) as that observed between previously injured and uninjured groups. We examined contiguous differences between groups across the entire stride for the kinematic data, and constrained our examination to the phase of stride occurring immediately prior to footstrike (equating to 30% of the entire stride) in the EMG data. This is due to the prestrike phase being the 'at risk' period for hamstring injury. In both cases we made comparison with 1,000,000 randomised datasets based on the original.

In order to ensure avoidance of Type 1 error, any comparisons where a statistical difference was reported, were further analysed by performing effect size calculations. The group difference in mean values for each timepoint across the normalised stride were

calculated and divided by the standard deviation of the control group data (i.e. the normal population) to give a Cohen's *d* value. The magnitude of the Cohen's *d* was interpreted as small (*d* = 0.2), medium (*d* = 0.5), and large (*d* = 0.8) as suggested tentatively by Cohen (125).

5.3 Results

5.3.1 Participant characteristics

There were no significant differences in anthropometric or sport specific characteristics between the groups at baseline ($p > 0.05$) therefore both groups were well matched with regard to age, height, weight and training exposure as well as team and playing position (Table 7). All previously injured participants had returned to full participation in sport and described a history consistent with an acute sprint related hamstring strain. Secondary confirmation of injury included contemporaneous diagnosis by a doctor/physiotherapist in 88.8% (8 of 9), positive findings on contemporaneous MRI in 44.4% (4 of 9) and the presence of post-injury bruising in 33.3% (3 of 9) of participants. The average time since injury occurrence was 11.17 months (SD = 9.2; range 1-23 months).

	Previously injured Group Mean (SD)	Control Group Mean (SD)	P values from Unpaired Student T tests
Age (years)	28.33 (5.24)	25 (5.39)	0.22
Height (m)	1.79 (0.058)	1.82 (0.049)	0.21
Weight (kg)	81.23 (6.56)	84.25 (6.99)	0.38
Playing exposure (hours/week)	7.3 (2.70)	7.5 (1.6)	0.84

Table 7: Participant Characteristics for the running study

5.3.2 EMG findings

There were significant contiguous reductions in EMG ratios in the BiFem:ipsiGluMax (maximum difference -12.5%, $p = 0.03$), BiFem:ipsiErecSpin (maximum difference -12.5% $p = 0.01$), BiFem:ipsiExtObl (maximum difference -23%, $p = 0.01$) and BiFem:contraRecFem (maximum difference -22%, $p = 0.02$) in the previously injured group. These reductions were confined to the late swing phase (see Figure 34). The bootstrapping analysis confirmed that these findings did not occur by chance alone (see *p* values outlined). These reductions in EMG ratios indicate that the biceps femoris on the injured side was relatively less active when related to the gluteus maximus, external oblique and erector spinae muscles on the

same side and the rectus femoris muscles on the opposite side. There were no differences in any of the other ratios calculated.

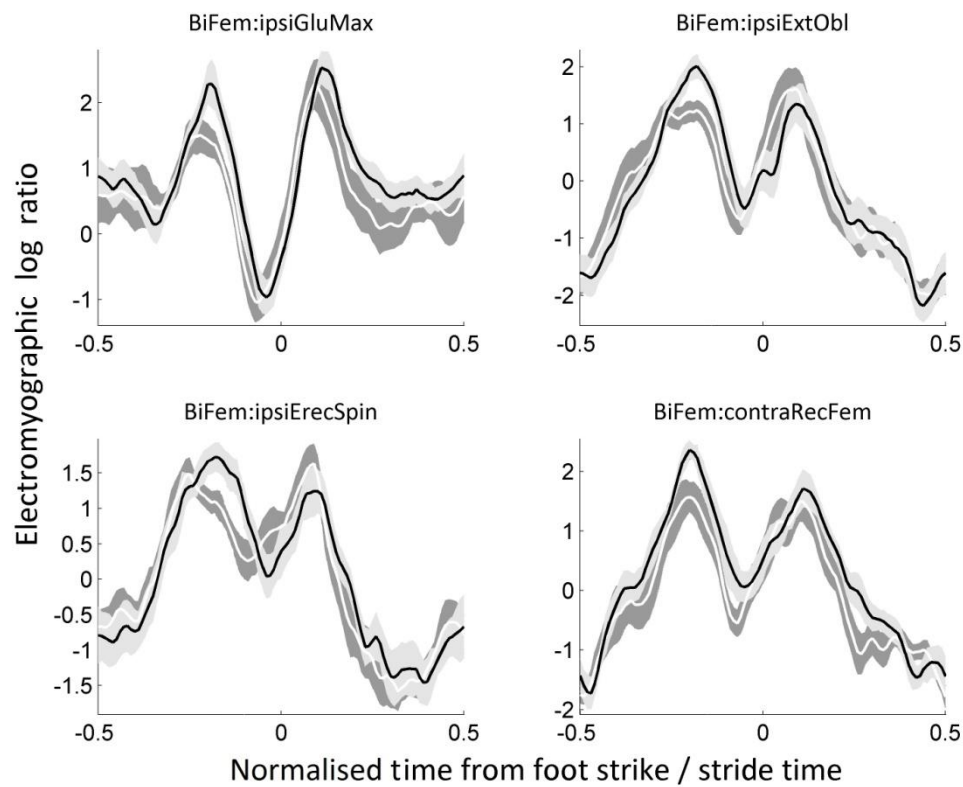


Figure 34: EMG ratio data for the Biceps Femoris:Ipsilateral Gluteus Maximus (BiFem:ipsiGluMax), Femoris:Ipsilateral Erector Spinae (BiFem:ipsiErecSpin), Biceps Femoris:Ipsilateral External Oblique (BiFem:ipsiExtObl) and Biceps Femoris:Contralateral Rectus Femoris (BiFem:contraRecFem). Mean log ratio values and standard errors are displayed for each group's stride – the zero mark on the x axes indicates foot contact. White line/Dark grey shading = previously injured group; black line/light grey shading = uninjured group. Plotted data has been smoothed by a factor of 3.

5.3.3 Kinematic findings

Asymmetrical movement patterns in the previously injured group were detected in the sagittal plane at the pelvis and hip. During the late swing, an increase in anterior tilt and hip flexion occurred on the injured side while reduced anterior tilt and hip flexion occurred on the uninjured side leading to maximum asymmetries of 4° ($p=0.02$) in pelvic and 8° ($p=0.01$) in hip movement. The degree of asymmetry detected within control participants was negligible (see Figure 35).

Transverse plane asymmetry of 6° (maximum) ($p=0.03$) was detected in the previously injured group in the knee joint during terminal swing and early stance phases. Marked

increases in medial knee rotation occurred on the injured limb and were largest in the early contact phase when compared to the uninjured participants (Figure 2).

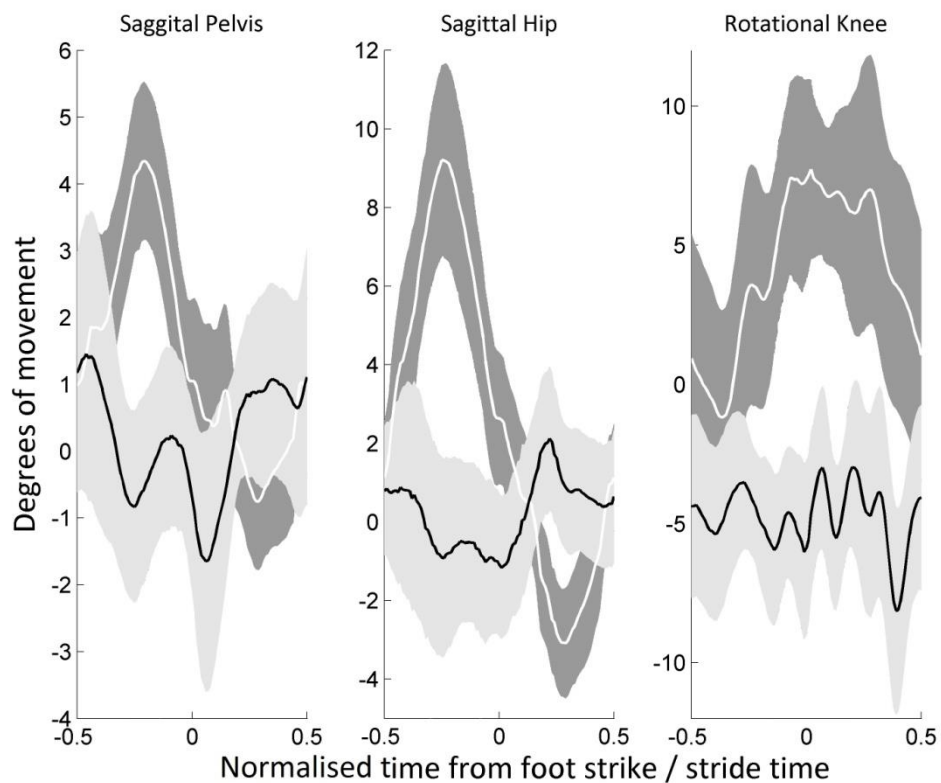


Figure 35: Kinematic symmetry data for pelvis and hip movement in the sagittal plane and knee movement in the rotational (i.e. transverse) plane during the average stride (101 points, foot contact defined as zero on the x-axes). Positive values on the pelvic graph = anterior tilt; positive values on the hip graph = flexion; positive values on the knee graph = medial rotation. Dark grey/white line = mean and standard error for previously injured group. White line/Dark grey shading = previously injured group; black line/light grey shading = uninjured group.

5.3.4 Effect size analysis

5.3.4.1 EMG findings

The Cohen's d values calculated across the entire stride for the BiFem:ipsiGluMax, BiFem:ipsiErecSpin, BiFem:ipsiExtObl and BiFem:contraRecFem are presented in Figure 36. Large effect sizes are noted in the late swing phase for all comparisons confirming the bootstrap analysis.

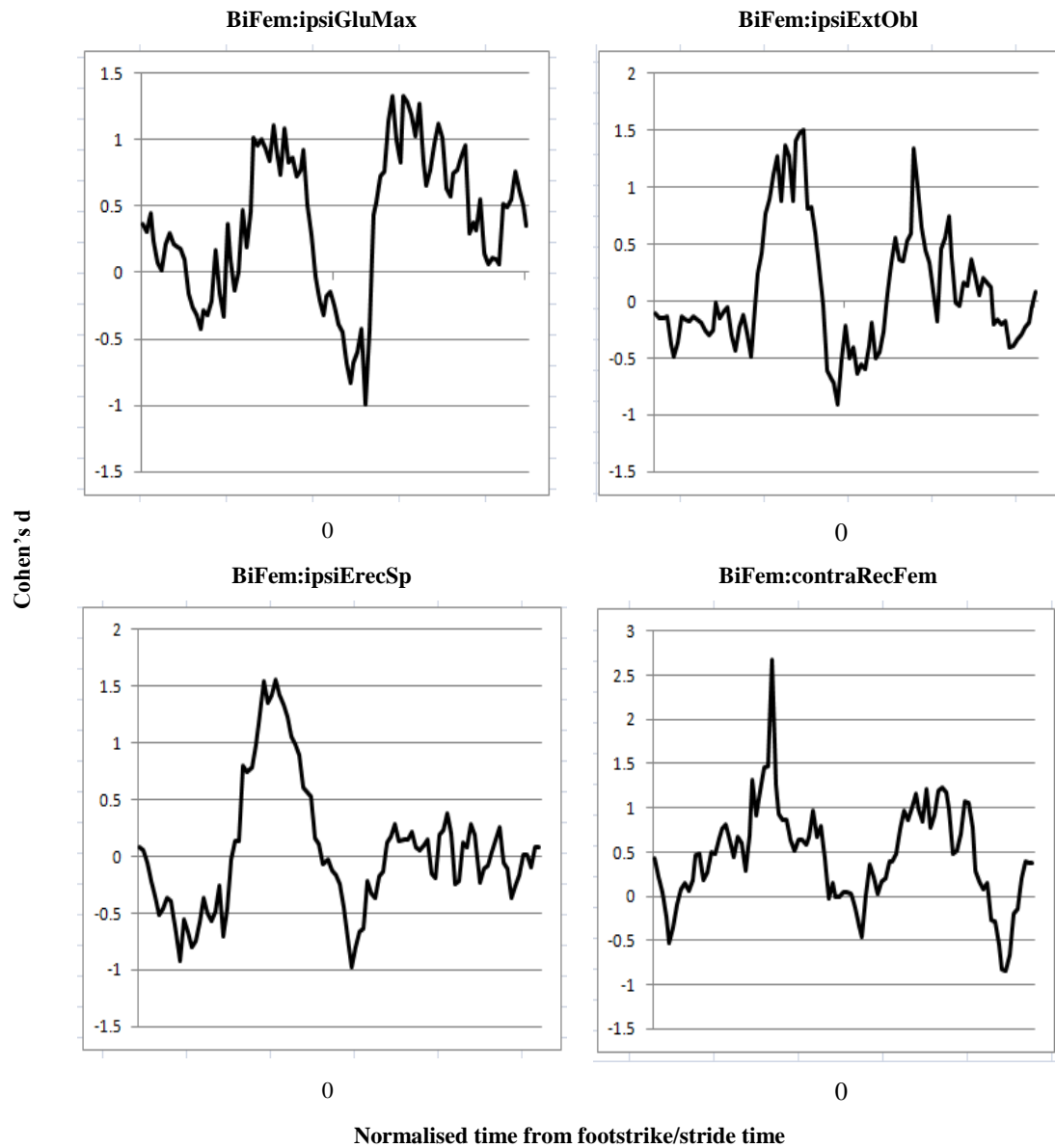


Figure 36: Effect sizes for EMG ratio data for the Biceps Femoris:Ipsilateral Gluteus Maximus (BiFem:ipsiGluMax), Femoris:Ipsilateral Erector Spinae (BiFem:ipsiErecSpin), Biceps Femoris:Ipsilateral External Oblique (BiFem:ipsiExtObl) and Biceps Femoris:Contralateral Rectus Femoris (BiFem:contraRecFem) during the average stride (101 points, foot contact defined as zero on the x-axes).

5.3.4.2 Kinematic findings

Data relating to effect sizes for significant kinematic outcomes are presented in Figure 37. There is a large effect noted ($d > 0.8$) when comparison is made between previously injured and control participants during the late swing phase for the pelvis and hip in the sagittal plane. The transverse plane knee comparison is also associated with a large effect through late swing and early stance.

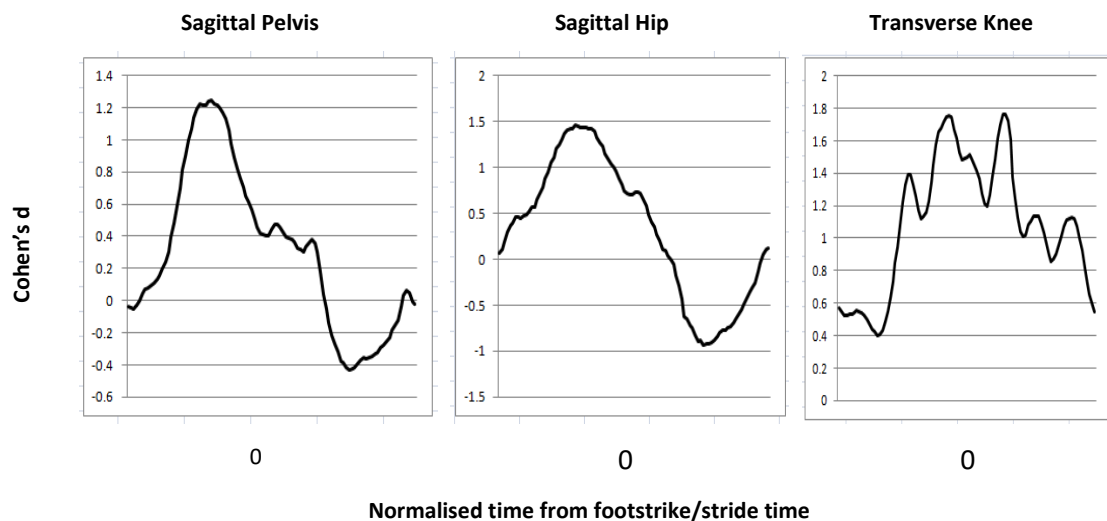


Figure 37: Effect size analysis for symmetry data for pelvis and hip movement in the sagittal plane and knee movement in the rotational (i.e. transverse) plane during the average stride (101 points, foot contact defined as zero on the x-axes).

5.4 Discussion

Participants with previous hamstring injury demonstrated significant reductions in EMG muscle activation ratios and asymmetries in pelvic and lower limb movement patterns during high speed running when compared to uninjured counterparts. We therefore rejected our null hypothesis and accepted the alternative that there were persistent biomechanical deficits in athletes with previous injury.

The combined movement asymmetries of increased anterior tilt, increased hip flexion and increased medial knee rotation on injured side are likely to place the long head of biceps femoris (BF) under increased strain to a greater extent than the medial hamstrings. Both the EMG differences and sagittal movement asymmetries are confined to the immediate pre-contact late swing phase of gait, a period when the BF longhead is thought to be at most risk of injury (72, 85).

The study demonstrated that side specific EMG and kinematic differences exist between athletes with and without previous hamstring injury during sprinting. Two previous studies examined running athletes with previous hamstring injury. The first, Lee et al (116) examined lower limb kinematics and found reduced hip flexion during terminal swing on the injured compared to the uninjured leg during submaximal sprinting. However Silder et al (177) failed to detect any between limb EMG or kinematic asymmetries in a group of individuals (n=18) during high speed treadmill running. The discrepancy between both these studies and this one may be explained by test population and methodological factors. Lee et al (116) and Silder et al (177) recruited a rather heterogeneous group in terms of age, sports, and injury mechanism with comparison made between injured and uninjured limbs only. The participants in the current study are homogenous in terms of anthropometrics, sporting exposure and injury mechanism (sprint related, contraction type), and the use of matched control participants ensure confounding factors such as exposure to altered training practices and sport specific biomechanics were optimally methodologically coded for.

Silder et al. (177) normalised EMG amplitudes to the mean values obtained during running trials. Although this is the recommended procedure for handling running EMG data (152), in studies where EMG signal amplitude may be fundamentally abnormal, as with previously injured muscle, the use of abnormal reference amplitudes may hinder the possibility of detecting real differences. We have attempted to avoid this methodological pitfall by expressing the findings as time-normalised EMG ratios, as used in previously published studies examining injured muscle (154).

Persistent post-injury hamstring specific functional asymmetries in athletes has previously been described in relation to measures of strength, angle of torque and EMG (173). Muscle morphological asymmetries have also been described (84). This study is the first to show clear and potentially injury inducing kinematic and muscle activity asymmetries during running after recovery and return to sport. Of particular importance is that both the changes in muscle activity and sagittal kinematic asymmetries were detected during terminal swing, the phase of gait linked to sprint related biceps femoris injury (48). The combined increases in hip flexion and anterior pelvic tilt on the previously injured limb can place the biceps femoris under increased strain. Notably, the kinematic changes are consistent with the EMG deficits detected.

Previous authors have suggested that increased anterior tilt may be associated with increased injury risk (24, 178). Some authors have postulated that the increased rates of hamstring injury seen in Afro-Caribbean and Aboriginal athletes may be explained by an increased tendency to adopt an anterior tilted pelvis (19). In this study, however, all participants, including those with increased anterior tilt on the injured limb were white Irish males. It has been suggested that a reduction of hamstring muscle activation may lead to a decrease in pelvic control (Sherry & Best 2004). It is therefore possible that the decrease in biceps femoris muscle activity may have contributed to the increased anterior pelvic tilt and hip flexion occurring during sprinting in this study.

An important observation is that the previously injured athletes ran with an apparently symmetrical gait pattern to visual inspection, which was significantly asymmetrical when measured with precision equipment. The retrospective nature of the study renders it impossible to determine if these biomechanical features existed prior to, or as a result of the injury, however these asymmetries were only present in the injured group. Chumanov et al. (46) highlighted the potential influence of lumbo-pelvic musculature on biceps femoris strain. In this study a relative increase in ipsiGluMax and ipsiExtObl combined with a relative decrease in BiFem activity were noted. It is acknowledged that we treated the biceps femoris as a single muscle and crosstalk between long and short heads may have influenced our data. Nevertheless, when considered in relation to the findings of Chumanov et al., (46) the changes in ipsiGluMax and ipsiExtObl activity could be considered 'protective' in nature as they are proposed to lead to reduced strain on the BiFem during terminal swing in sprinting. However, in addition to these findings, a relative increase in contaRecFem and ipsiErecSpin were also noted, factors which according to Chumanov et al. (46) would lead to increase strain during terminal swing. This altered muscle activity may represent an adaptive process, a failure to adapt to BiFem specific dysfunction or persistent post-injury neuroinhibition (61). The relative decrease in hamstring activity and associated biomechanical changes in late swing, when the greatest hamstring activation is needed, may be indicative of such inhibition.

The apparent loss of rotational control at the knee joint on impact (increased medial rotation) is an important, albeit unexpected, finding. Increased medial rotation at the knee is a mechanism closely associated with meniscal and anterior cruciate ligament injury (179). The association between biceps femoris functional inhibition and associated articular injury

risk has not been established, however previous injury to a limb is associated with increased risk of further injury to that limb (180). Given the anatomical arrangement of the medial (semitendinosus and semimebranosus) hamstrings and the biceps femoris, some have postulated that these muscles may act to control knee rotation (181). Should the biceps femoris be limited in its capacity to control rotation, this could result in increased medial rotation on foot contact. Although it is acknowledged that there is some risk of measurement error when calculating knee rotation using the methods employed in this study, the combination of EMG changes and kinematic asymmetries appears to indicate such a loss of rotation control about the knee joint. Further research should aim to confirm this apparent association as this may highlight potential articular injury risk following hamstring injury.

It is acknowledged that we did not collect EMG from the contralateral biceps femoris or the medial hamstrings on both limbs. Future research could explore if bilateral and/or medial EMG alterations exist in this population in order to determine if the changes we measured manifest across both limbs (i.e. via central mechanisms) or are confined to the side of injury. This may highlight useful rehabilitation strategies where targeted loading may induce specific adaptations with the neural and or muscular tissue and reverse the deficits detected (182). A key limitation of this study is the retrospective nature of recruitment and testing. We therefore cannot determine if our findings existed in advance of, or as a result of the injury. Additionally, although we employed clear inclusion criteria to target sprint related structural hamstring injury, only 4 participants had confirmatory imaging completed at the time of injury. These images were not viewed and the exact location of the previous injured could not therefore be confirmed. Although the lack of diagnostic imaging is not unusual in sport, we relied on description of injury mechanisms and effect to determine a history of injury, confirmed in nearly all cases by a professional examination. It was not possible to measure pre-injury performance capacity in term of maximal sprint speed or maximal knee flexor strength in the previously injured group. Therefore it was not possible to match control participants with respect to these measures and therefore some discrepancies may exist between the study groups. Nevertheless, subject pairs were recruited from the same team and level and therefore were likely to have similar performance capacities. Instrumented strength analysis was not included in this study. It is acknowledged that this would add value as a separate outcome measure. Finally, we

accept that subject numbers are small, limited to one sporting population and that we did not attempt to subgroup according to time since injury. Some of the subjects who had sustained a recent hamstring injury may have has residual low level weakness which was not detected during screening. We therefore highlight the need for further research to address these issues and recommend caution when attempting to generalise these findings across all sporting populations.

This study indicates that important, potentially injurious and detectable biomechanical features are present in athletes with previously injured hamstrings during running. These features were present up to two years after injury and were not associated with any subjective or sport limiting symptoms. Addressing such deficits are a research and clinical priority. Further research should confirm these findings and explore if such asymmetries can be reduced within rehabilitation. Whether such an effect would reduce the high levels of recurrence seen in hamstring injury is yet to be determined.

5.5 Clinical impact

Hamstring injury, specifically related to the biceps femoris muscle, remains a major problem in sprint based sport. Recent research (66) highlights that hamstring injury is the most prevalent injury (24.1%) seen in track and field sport, as it is in soccer (2), Gaelic games (3, 4), Australian football (8) and rugby (5). Furthermore, the leading risk factor for hamstring injury is previous injury (22). This study shows a clear association between previous hamstring injury and persistent alterations in biceps femoris muscle activity and lower limb movement control even following successful return to sport. The altered kinematics place the hamstring at increased length during the critical late swing phase, while the inhibition of biceps femoris muscle activity represent a key target for rehabilitation. It may be that addressing such deficits reduces recurrence – a key rehabilitation goal, and therefore a topic for future work.

5.6 Conclusion

Significant EMG and kinematic changes were identified during running in athletes with previous hamstring injury. These findings indicate reduced biceps femoris associated muscle activity during the late swing phase. Significant late swing phase asymmetries in sagittal pelvic and hip movement were detected on the injured limb, likely to place the

hamstring complex at increased length during this crucial phase of running gait. Furthermore, previously injured limbs are characterised by increased medial knee rotation on foot contact. These findings constitute biomechanical risk factors for hamstring re-injury which should be considered when designing injury prevention and rehabilitation strategies.

6 Observational Study: Nordics (eccentric loading)

Spatial activation of the hamstrings during the Nordic loading exercise: a case control study of previously injured athletes.

6.1 Introduction

Hamstring injuries are common in sports which involve sprinting (2-4, 6, 9, 11, 12, 66, 67, 82, 183). Despite advances in understanding the mechanisms of this injury, and developments in post-injury rehabilitation strategies, previous injury remains the leading risk factor for future injury (11, 20). With re-injury rates remaining persistently high, there is a need for further research into the biomechanical characteristics of athletes following hamstring injury. Such research may highlight key characteristics of post-injury hamstring related dysfunction and provide primary data for further research. Specifically, determining a causal relationship between a hamstring related biomechanical factors and an increased risk future injury will provide a key target for enhanced rehabilitation approaches. The ability to screen for such characteristics, should causative risk be established, will be invaluable.

Research exploring EMG activity in previously injured hamstrings is limited. As reported in Chapter 3 of this thesis, six studies have investigated hamstring EMG in previously injured athletes. Three of these measured muscle activity during isokinetic contractions (69, 70, 108), two during running (103, 119) and one during a double to single leg support movement (109). Very limited evidence indicates reduced amplitude biceps femoris activity during eccentric loading in previously injured athletes compared to their contralateral limbs (69, 70) and compared to control limbs (108). There is also some very limited evidence of reduced biceps femoris related EMG ratio activity during terminal swing in running (103) (see also Chapter 5). In contrast, there is also very limited evidence of no change in EMG amplitude symmetry during running (119) or differences in hamstring median frequency outcomes between previously injured and control subjects during isokinetic loading (69). The level of evidence available and the contrasting findings in these studies mean that the value of EMG in determining post-injury hamstring dysfunction is not well established.

Standard bipolar EMG provides information about the motor unit action potentials generated within range of the skin sensors (130, 158). EMG activity appears to be non-uniform across muscle bellies. Different regions within a muscle contribute in differing ways during a contraction. Factors such as the distribution of muscle fibre types, the architecture of the underlying muscle fibres, the location of the motor end points, the distribution of motor units and the nature of the demands being placed upon the muscle, will all have a bearing on the intensity and location of the myoelectric signal (158-162). Several studies have explored the regional distribution of EMG activity using low density, high surface area arrays of sensors with outcomes related to fatigue and delayed-onset muscle soreness predominating (159-163). Despite extensive searching of the literature, no studies were identified which explored spatial activity following an acute structural muscle injury.

The research outlined in the previous paragraph, as well as the methods used in this chapter are separate to the larger body of research where high density electrode arrays are placed over a small area. This alternative approach is typically concerned with measuring the propagation of motor unit action potentials (MUAP) across a small area of muscle (134). Our methods are designed to measure spatial activation patterns across the entire muscle belly, with lower density arrays spread over larger areas. We do not attempt to measure, nor present data related to MUAP propagation.

One full paper and two abstract only publications report low density, high surface area EMG data from the hamstring muscles. All investigated a healthy and not previously injured population. Watanabe *et al* (184) found that the biceps femoris average rectified EMG amplitude was statistically uniform across the length of the muscle (in contrast to the data presented above) during a combined an isometric knee flexion and hip extension contraction. They measured activity along the length of the biceps femoris belly using a 20 electrode linear array. Morrissey *et al* (185) and Sakthibalan *et al* (186) present abstracts from the same dataset, where they report consistent patterns of EMG activity across the hamstring region during various functional tasks including walking, running and kicking. These authors used 16 channel bipolar arrays covering the posterior thigh.

The apparent consistency in patterns of muscle activity across the hamstrings, highlights a potential application in screening previously injured athletes. Recent evidence points to

persistent post-injury neuroinhibition as potentially underlying the elevated re-injury risk seen with hamstring injury. Evidence for such inhibition includes reduced relaxation times on functional MRI following maximal effort exercise (68), and altered slow velocity eccentric knee flexor torque profiles of previously injured athletes (69, 70, 105, 115). However, functional MRI remains relatively inaccessible and expensive to the wider public. Furthermore, as outlined in Chapter 3 of this thesis, the evidence supporting deficits in slow eccentric strength is limited to lower and moderate quality research.

Opar *et al* and Timmins *et al* provide some evidence that an instrumented version of the Nordic loading exercise may offer screening potential for hamstring strength deficits (65, 71, 121). The 'Nordic' is a high demand bodyweight loading exercise developed specifically to deliver eccentric strengthening stimulus to the hamstrings. The exercise is initiated in a kneeling position with the ankles tethered. The subject maintains extended hips and an erect trunk, while lowering their chest to the floor via slow extension of both knees (64). Furthermore, the Nordic appears to reduce re-injury risk when performed as a progressed loading protocol during pre-season training in footballers (64).

The aims of this study are to explore the potential application of low density multichannel EMG in detecting post-injury neuromuscular inhibition in the hamstrings during a maximal effort eccentric loading activity (the 'Nordic'). The null hypothesis is that there would be no difference between previously injured and uninjured groups with respect to the magnitude of EMG activity at the time of peak force or peak amplitude activity during a Nordic loading exercise. The alternative hypothesis is that there would or would not be evidence of persistent hamstring inhibition and that intra-subject and/or limb EMG asymmetry would or would not reflect this inhibition.

6.2 Methods

6.2.1 Participants

Ethical approval for this study was obtained through the Queen Mary University of London human research ethics committee and all participants provided written informed consent before participation. Sixteen football code athletes (soccer and rugby) from both professional and university level football volunteered to take part. Eight had a history of acute onset sprint related hamstring injury within 36 months of participation. Control

subjects were matched for sporting level, age, height and training exposure where possible in order to minimise the effect of confounding variables.

The previously injured group comprised of eight subjects (one further subject was excluded). All had a history of unilateral sprint related hamstring injury within three years of participation in this study and all had since returned to full participation in sport. Specifically, all reported: (a) a history of sudden onset posterior thigh pain which occurred during high speed running, (b) pain/discomfort localised to the hamstring region, (c) pain/discomfort on active contraction and running for a minimum of 48 hours post-injury, (d) an inability to participate in normal sporting activity for a minimum of 48 hours (127). As a secondary means of injury confirmation, subjects were questioned regarding the presence of post-injury bruising and whether they underwent contemporaneous assessment by a registered physiotherapist/medical professional and/or confirmatory imaging. Time since injury and time out of sport data were recorded. In addition, information about prior exposure to the Nordic loading exercise was elicited.

Subjects were excluded if they had not returned to full participation in sports at the time of testing. Furthermore, subjects were excluded if they presented with any signs of residual hamstring muscle, articular (sacro-iliac and lumbar spine) or neural dynamic (sciatic) dysfunction. Specifically, all subjects were assessed by an experienced musculoskeletal physiotherapist for hamstring weakness (using manual muscle testing) (126), pain on palpation of the hamstring muscles, articular dysfunction using segmental articular mobilisation of the lumbar spine, sacral thrust, compression and distraction tests, and neural dynamic dysfunction using both a slump and straight leg raise test (127). Subjects were also excluded if they had a history of lower limb surgery within two years of testing, had positive findings on the Physical Activity Readiness Questionnaire (175). Control subjects were excluded if they had ever sustained a suspected hamstring injury.

6.2.2 Experimental Process

All subjects attended for one data collection session at the Human Performance Laboratory at Queen Mary University of London. Measures of height and weight were calculated using a Leicester height measure and calibrated scales. Sports and injury details were collected via direct questioning. Each subject underwent a clinical examination (as outlined above) by a musculoskeletal physiotherapist with over five year's clinical experience.

Subjects were asked to perform a Nordic loading exercise (the 'Nordic') while simultaneous EMG and force data was collected. To complete the 'Nordic', the subject begins in an upright kneeling position on a cushioned surface. His/her ankles are fixed or held in place. With their arms crossed over their chest, the subject lowers their trunk towards to floor, without flexing at the hip or spine. In this way, it is proposed that the majority of the load is transmitted through the hamstrings as they eccentrically lower the trunk to the floor. The subject slowly leans forward until they can no longer maintain control. At this point the hamstrings reach their maximum ability and the subject rapidly falls forward using the hands to break the contact with the floor in front. Often the subject returns to an upright position (the concentric phase) with minimal assistance from the upper limbs. In this study, we elected to focus our analysis on the eccentric phase (i.e. the lowering phase) only as it is thought that this is the primary driver of recovery when the 'Nordic' is employed during rehabilitation (187).

6.2.2.1 Multichannel EMG

In order to explore the spatial muscle activation patterns, four linear arrays of unipolar EMG sensors was fixed to the skin over the hamstring muscles. The skin was prepared according to SENIAM guidelines, being shaved, abraded and cleaned with an alcohol wipe in order to maximise adhesion and minimise impedance (136). On each leg, 32 pre-gelled sensors were affixed to the skin in 4 longitudinal arrays of 8 sensors. Specifically, the mid-point of the biceps femoris (combined short and long head) was identified as being 50% along a line from the ischial tuberosity to the lateral epicondyle of the tibia. The first column of sensors was affixed to the skin immediately lateral to this line with the midpoint of the muscle transecting the midpoint of the column i.e. four sensors lay proximal (and slightly lateral) to the mid-point and four lay distal. The next column was affixed to the skin immediately medial to the biceps femoris line. The same procedure was used on the medial muscle bellies, with the two columns of eight sensors affixed either side of a line drawn from the ischial tuberosity to the medial tibial epicondyle with the midpoint of the column lying over the midpoint of the line. The silver-silver chloride surfaces of the sensors were covered with conductive gel using a syringe and the sensor affixed to the skin using double sided tape perforated with circles of 5mm diameter at 20mm intervals while the subject lay in a supine position with the knee slightly flexed. Adhesive tape (MediTrans, Fleming Medical Ltd., Ireland) was additionally applied over the arrays to ensure maximal

fixation. A ground electrode was affixed to the skin over the calcaneus. This arrangement is illustrated in Figure 38. Data was collected at 2048hz using a 64 channel EMG system (REFA64, TMSI, Netherlands) and stored directly onto a harddrive for offline analysis.

6.2.2.2 Force measures

Simultaneous measures of force were collected using the same hardware as the EMG data. Two independent load cells (TSA Alloy Steel S Type, Coventry Scales Company, UK) were attached to the subjects' left and right ankles using heavy duty ankle cuffs and climbing carabinas. The opposite end of each load cell was orientated at a right angle to the longitudinal axis of the lower leg and attached to a metal d-ring which, in turn, was screwed into to a 20mm marine plywood board. This board extended under the platform upon which the subject was kneeling and provided an inflexible anchor for measuring forces through the ankle during the loading exercise. This device was fabricated with particular reference to the device designed by Opar et al (121) and is illustrated in Figure 39.

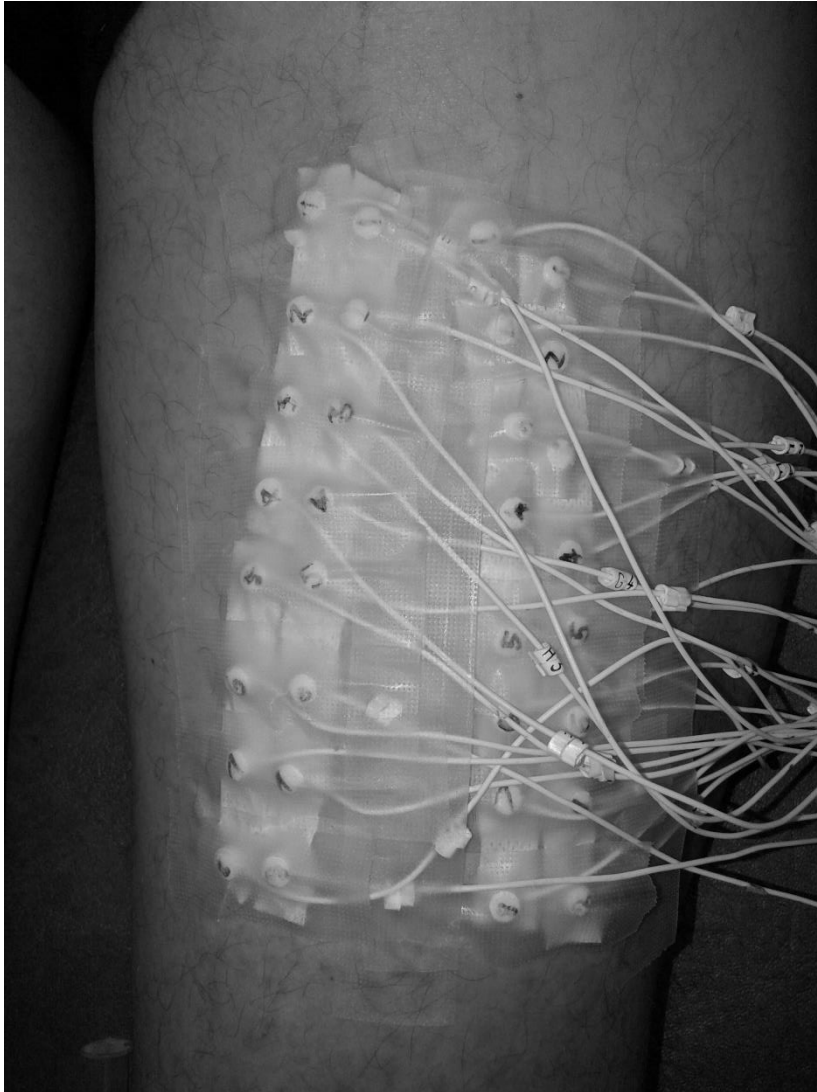


Figure 38: Multichannel EMG arrangement

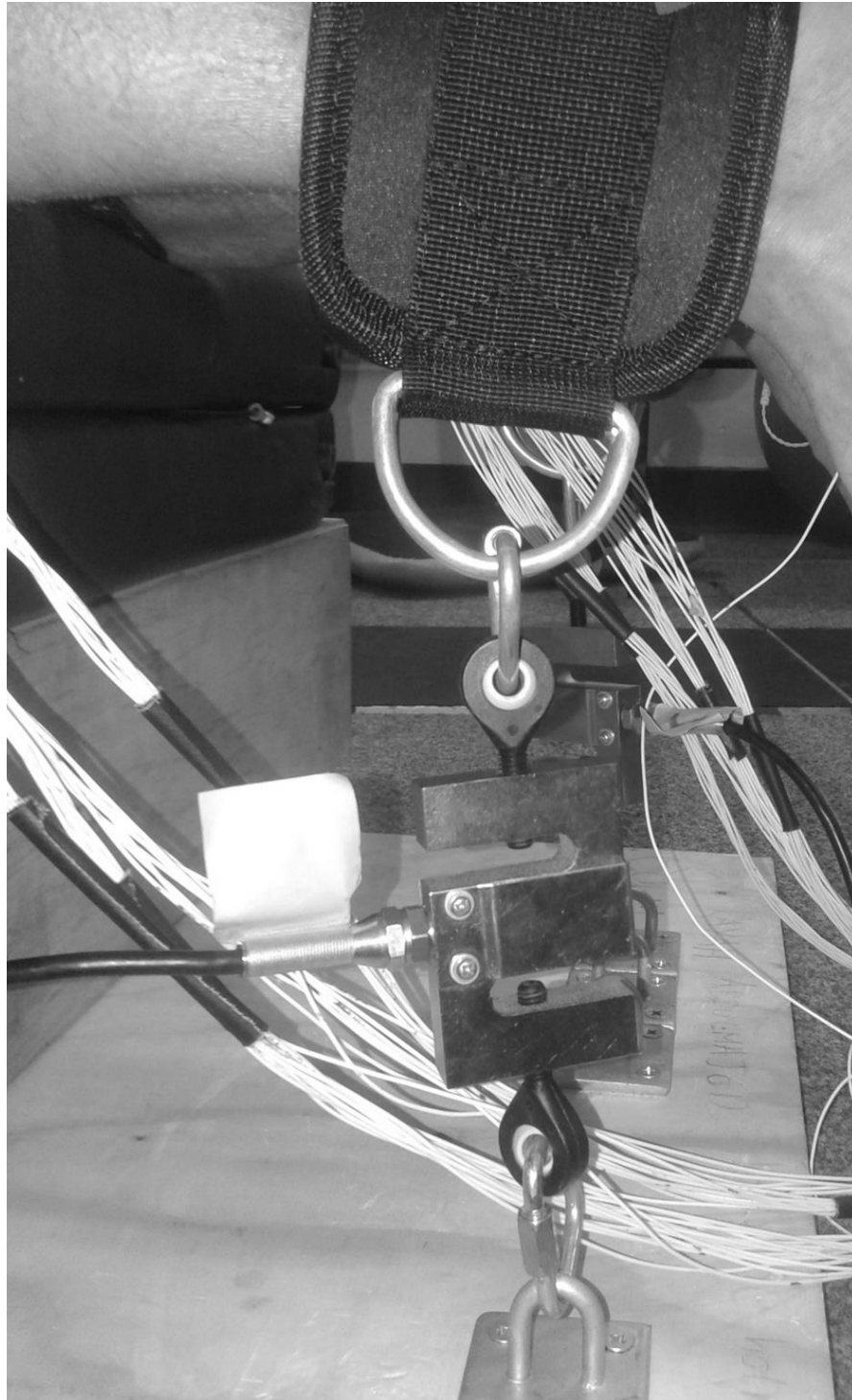


Figure 39: Illustration of load cell set-up

6.2.3 Data analysis

All data was analysed offline using a custom written Matlab programme. The voltage output of the load cells was converted to kilogram weight using a multiplication factor

derived from calibration tests which were performed separately (see Methods development 4.4.3.2). The time point and magnitude of peak force was visually identified and marked for each subject limb. Average peak force was calculated across three trials for all participants in each group.

EMG signals were band pass filtered at 10-500hz in order to remove frequencies unrelated to muscle activity as per international guidelines (136, 164). Furthermore, a notch filter about 50hz was applied to ensure removal of ambient electrical interference. Individual signals were examined for quality. Data was removed from further analysis when motion artefact adversely affected signal quality (low frequency, high amplitude signal) or where data was missing due to detached/damaged sensors. Finally, the signals were full wave rectified.

Extrapolation of clinically meaningful EMG data was performed using four separate methods outlined below. Inter-group comparisons were made between previously injured and uninjured limbs of previously injured subjects. In addition, intra-group comparisons were made between the previously injured and uninjured limbs of the previously injured group and the dominant limbs of the control group. Dominance was defined according to preferred kicking limb (188).

6.2.3.1 Normalised EMG signal at time of maximal force

The filtered EMG data was rectified and smoothed using the Root-Mean-Square (RMS) method with a 50hz window applied. The EMG signal was found to lack a clear onset and finish time owing to the subjects performing isometric contractions at the beginning and end of the Nordic loading exercise. RMS EMG data corresponding to this period of loading associated with the performance of the Nordic loading exercise was extracted for analysis. RMS signals for each sensor were normalised to the mean RMS amplitude during the loading period. Peak normalised RMS amplitude was then calculated for each channel. This approach conforms to standard methods for surface EMG signal processing (130, 152).

6.2.3.2 Channel by channel contribution to maximal EMG activity

The aim of the second analysis was to explore the contribution of each channel to overall maximal hamstring EMG signal and thus provide a spatial overview of hamstring muscle activity during the 'Nordic'. A novel procedure was developed to achieve this as follows. The mean activity across all 32 channels on each leg was calculated and smoothed by a

factor of 500 (i.e. an interpolation was performed at 512hz intervals or approximately every 25ms). Maximal activity was defined as all timepoints within the top 20% of the total and smoothed mean value. The activity for each channel occurring during this timeframe was extracted. Finally, the filtered and rectified amplitude data for each channel was expressed as a ratio of the total activity occurring across all channels, thus providing a ratio of activity at each sensor.

6.2.3.3 Integrated EMG activity (iEMG)

As the duration of loading varied between participants, the RMS EMG data was time normalised by interpolating all data to the number of samples in the longest duration sample. This enabled calculation of iEMG values without confounding influence from contraction duration. iEMG calculations were performed on the time normalised RMS EMG data using the trapezoidal method to calculate the area under the curve of the signal for each channel.

6.2.3.4 Frequency Analysis

The median frequency for the EMG signal of each channel was calculated. The filtered, unrectified signal was extracted for the duration of the Nordic loading exercise, using the force onset and offload times as described above. A discrete Fourier transform was performed across the entire signal (i.e. at 2048hz) to extract the frequency components. The median frequency was subsequently calculated for each channel.

6.2.4 Statistical Analysis

Close participant matching was insured by conducting student t-tests on parametric variables (age, height, weight, playing exposure) and Mann Whitney U testing on categorical data (sporting level, playing position). The distribution of force data was analysed for normality. In order to ascertain levels of peak force, comparisons were made between the previously injured limb and uninjured limb of the previously injured group and the dominant and non-dominant limb of the control group using independent student t-tests. Furthermore, the magnitude of asymmetry in peak force values was calculated for each subject by subtracting the peak force generated by the uninjured limb/non-dominant limb in controls, from that generated by the previously injured limb/dominant limb in controls. The product of this calculation was compared between groups using independent

sample Student T tests. Given the use of multiple t-tests, a Bonferroni correction was applied to any significant findings to avoid Type 1 error.

EMG data was explored for intergroup difference using a three-way ANOVA. Group (previously injured and comparator), lateral-medial position of sensors (corresponding nominally to lateral biceps femoris, medial biceps femoris, semitendinosus and semimebranosus) and longitudinal position of sensors (8 positions at 20mm intervals arranged proximal to distal) were inputted as fixed factors and the EMG outcome in question as the dependent variable. Where significant differences were identified, pairwise comparisons were made by visually inspecting graphs of the estimated marginal means according to group. Separate analyses were completed for normalised EMG, EMG contribution, integrated EMG and median frequency values respectively. As data derived from EMG values are naturally skewed in an inverse Gaussian manner (due to a zero mean), an aligned rank transform was conducted on datasets to reduce the risk of Type 1 and Type 2 error (167-169). This procedure allows for factorial analysis of data which has been aligned, ranked and rescaled according to the factor (or factor interaction) being tested. Secondary ANOVA tests were performed on the ranked and rescaled datasets. The non-parametric Mann Whitey U test was avoided as this test is unable to measure factorial interactions and subdivision of the dataset may increase the risk of Type 1 error.

In order to ensure avoidance of Type 1 error and ensure sufficient powering, any comparisons where a statistical difference was reported, were further analysed by performing effect size calculations. The group estimated marginal mean values were used to give a Cohen's d value. The magnitude of the Cohen's d was interpreted as small ($d = 0.2$), medium ($d = 0.5$), and large ($d = 0.8$) as suggested tentatively by Cohen (125).

In order to examine the influence of dominance, we made comparisons between the dominant and non-dominant limbs of the control group. Primary inter-group comparisons were made between the previously injured limb and the dominant limb of controls. Where a significant difference between control subject limbs was detected, a further comparison was made with the non-dominant limb.

6.3 Results

6.3.1 Participant Characteristics

Cases were closely matched for anthropometric, training level and training exposure (see Table 8 and Table 9). One subject in each group was female. All previously injured subjects reported a subjective history consistent with a sprint related hamstring injury within the previous three years. In addition, all reported a resolved history of pain on resisted knee flexion and stretching of the hamstring at the time of injury. The mean time since injury was 13.1 months (SD 10.02, Range 2-36 months). Time absent from sport was reported on average as 17.9 days (SD 9.8, Range 7-30 days). Injury was confirmed by medical examination in 62.5% of cases. No participants reported having confirmatory MRI nor did any experience bruising after injury. Five participants in the previously injured group and four in the control group were professional association footballers, all participating in the Football League (Division 1 and 2). The remainder played university level amateur football. One professional footballer was excluded from the previously injured group as he displayed symptoms of neuro-dynamic dysfunction (positive slump and straight leg raise test) rather than muscle injury.

	Previously injured Group Mean (SD)	Control Group Mean (SD)	P values
Age (years)	19.6 (1.58)	18.5 (0.76)	0.25
Height (m)	1.78 (0.09)	1.75 (0.09)	0.49
Weight (kg)	72.8 (8.9)	67.5 (9.6)	0.27
Playing exposure (hours/week)	7.4 (3.2)	6.7 (4.3)	0.70

Table 8: Participant characteristics (scalar). All data is reported as group mean and standard deviation (SD).

	U Score	Significance
Playing level (non-elite/elite)	28	0.52
Playing position (Goal, defense, midfield/attack)	27	0.63

Table 9: Participant characteristics (categorical). U values and significance from Mann Whitney U tests are reported.

6.3.2 Force data

Force data was found to be normally distributed. There were no significant differences in peak force detected between previously injured (28.9 (5.7) kg) and uninjured limbs (29.5 (8) kg) in the previously injured group ($t=-0.15$, $p=0.88$) (see Figure 40), nor between dominant (23.6 (8.2) kg) and non-dominant (22.7 (7.5) kg) limbs in the control group ($t=0.23$, $p=0.82$) (see Figure 41). Furthermore, the magnitude of asymmetry between limbs was not statistically different between groups (previous injured 0.5 (4.5) kg; control 0.9 (2.2); $t=-0.8$, $p=0.43$) (see Figure 42). Thus, no deficits in force generating capacity were detected either within or between previously injured and control groups.

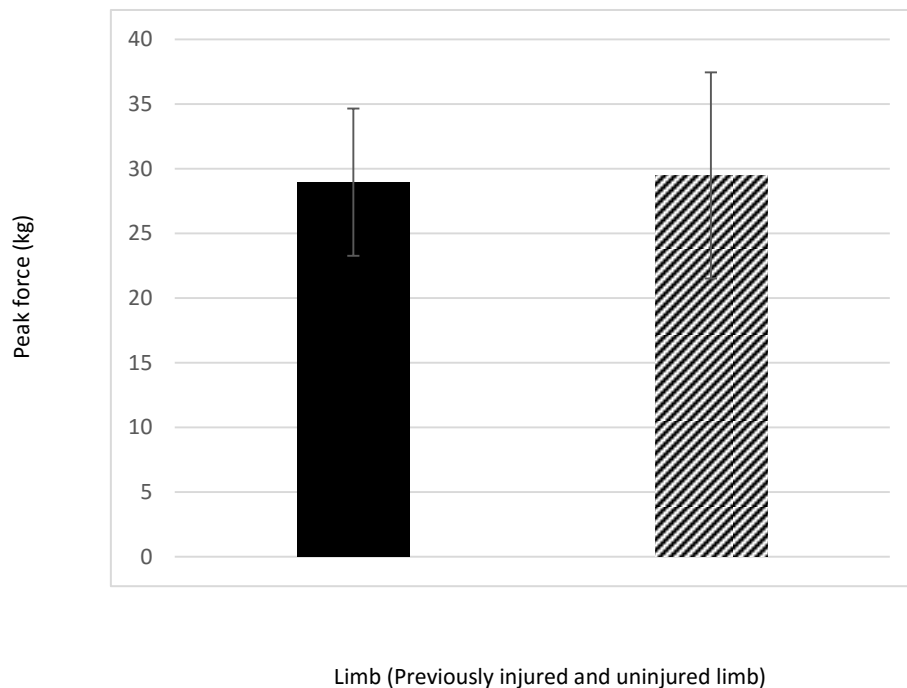


Figure 40: Average peak force (kg weight) generated by the previously injured limb (black) and uninjured limb (shaded) in the previously injured group. Error bar = 1SD

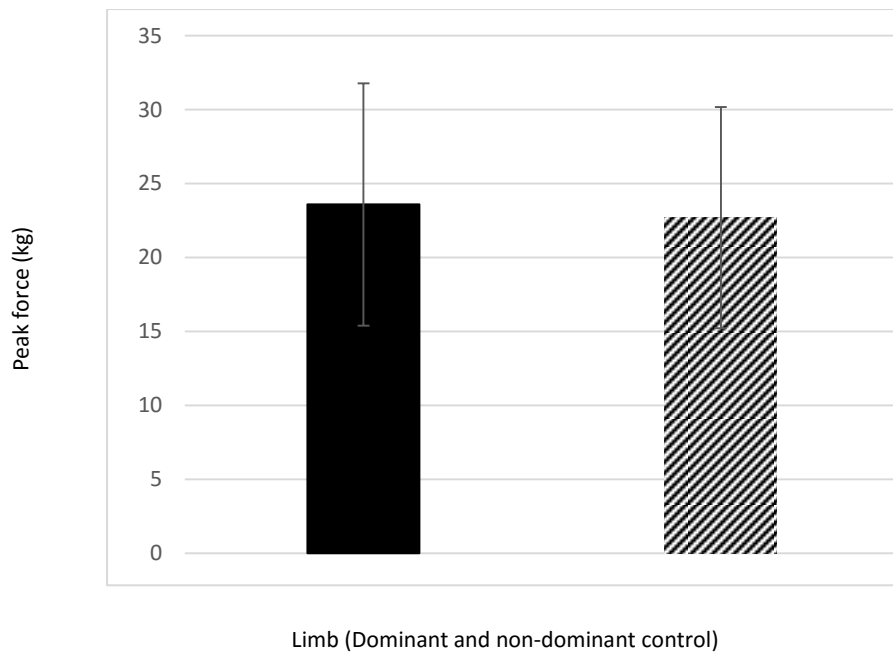


Figure 41: Average peak force (kg weight) generated by the dominant limb (black) and non-dominant limb (shaded) in the control group. Error bar = 1SD

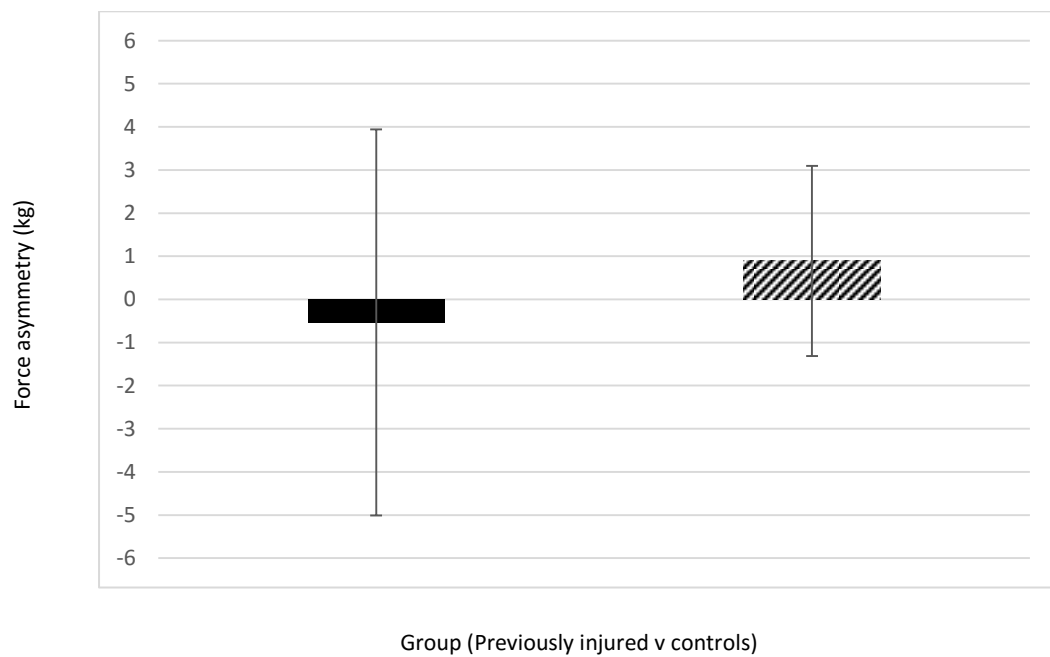


Figure 42: Force asymmetry between limbs in previously injured (black) and control (shaded) groups. Error bar = 1SD

6.3.3 EMG activity (normalised to mean) during loading

There was no statistical difference in peak normalised RMS EMG between previously injured and uninjured limbs in previously injured subjects. This finding was confirmed using three-way factorial interaction analysis (group x vertical location x horizontal location of the sensor, $F=0.26$ $p=1.00$) as well as both two-way interactions (group x vertical location, $F=0.991$, $p=0.17$; group x horizontal location, $F=0.46$, $p=0.71$). Secondary analysis using aligned rank data confirmed that there was no significant difference between groups for the three-way factorial analysis ($F=0.26$, $p=1.00$) or for either two-way interaction (group x vertical location, $F=0.21$, $p=0.98$; group x horizontal location, $F=0.47$, $p=0.70$) (see Figure 43).

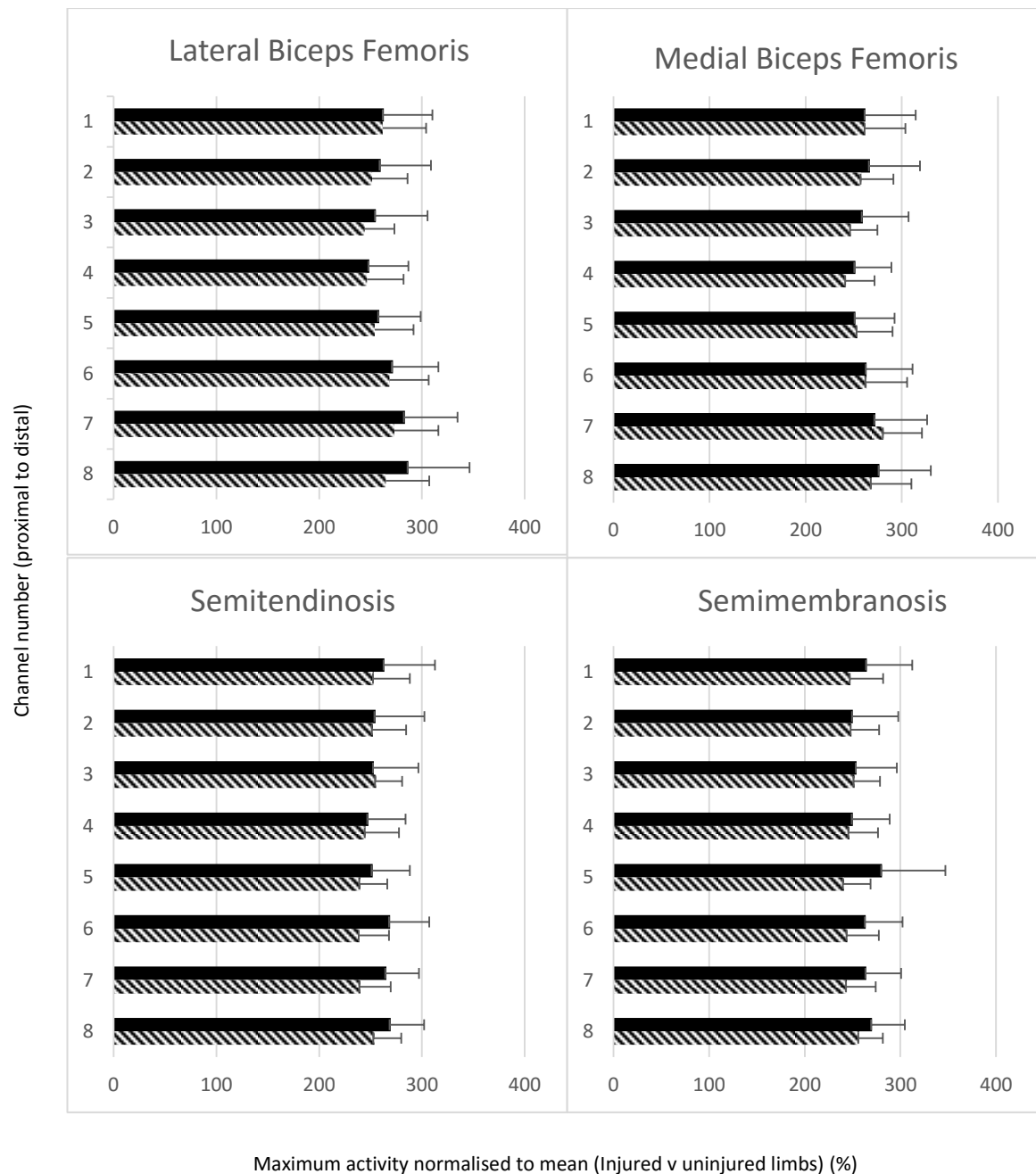


Figure 43: Maximum EMG amplitude (normalised to mean amplitude) during the period of maximal force production for each array of eight sensors. Channel numbers are aligned vertically (proximal to distal). Subplots indicate values for each of the horizontal locations nominally indicated by corresponding muscle (medial to lateral). Black bar= previously injured limb; Hatched bar = uninjured limb (in previously injured group). Error bar = one standard deviation.

When comparisons are made between the previously injured limb and dominant control limb, no significant differences are noted in peak normalised EMG activity for the three-way factorial interaction (group x vertical location x horizontal location, $F=0.22$, $p=1.00$) (see Figure 44) or either two-way factorial interaction (group x vertical location, $F=0.04$,

p=1.00; group x horizontal location, F=0.40, p=0.76). Analysis of rank transformed data rescaled according to factorial interactions also shows no significant difference for the three-way interaction (F=0.22, p=1.00) nor either two-way analysis (group x vertical location, F=0.04, p=1.00; group x horizontal location, F=0.39, p=0.76) (see Figure 44).

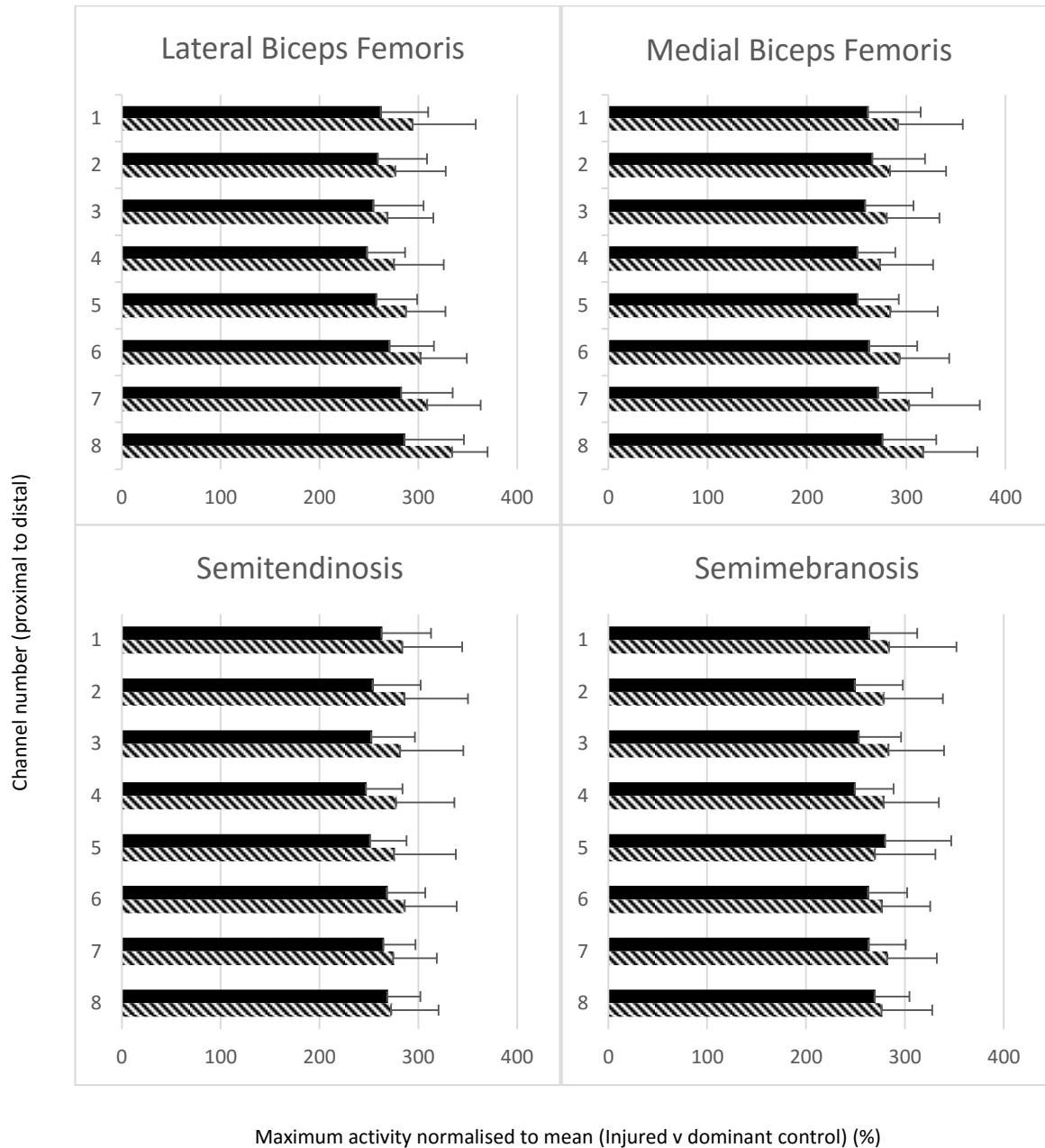


Figure 44: Maximum electromyographic amplitude (normalised to mean amplitude) during the period of maximal force production for each array of eight sensors. Channel numbers are aligned vertically (proximal to distal). Subplots indicate values for each of the horizontal locations nominally indicated by corresponding muscle (medial to lateral). Black bar= previously injured limb; Hatched bar = dominant limb (in control group). Error bar = one standard deviation.

6.3.4 Channel contribution to maximal EMG signal

There was no difference in the pattern of maximal activation between the previously injured hamstring and uninjured hamstring of previously injured athletes when the amplitude of each channel is considered as a ratio of contribution to overall maximal activity (three-way ANOVA for group x vertical location x horizontal location, $F=0.12$, $p=1.00$) (see Figure 45). Furthermore, when analysis is limited to either the vertical or horizontal location of the sensor, no significant differences are noted (two-way ANOVA for group x vertical location, $F=0.67$, $p=0.7$; group x horizontal location, $F=0.03$, $p=0.99$). Secondary analysis following transformation to rank order and realignment according to factorial interactions confirms no significant differences between groups for the three-way (group x vertical x horizontal location, $F=0.12$, $p=1.00$) nor either of the two-way interactions (group x vertical location, $F=0.81$, $p=0.58$; group x horizontal location, $F=0.03$, $p=0.99$) (see Figure 45).

When comparison is made between previously injured limbs and the dominant limbs of control subjects, no significant difference between limbs is noted when both horizontal and vertical location of the sensor is considered together (three-way ANOVA for group x vertical location x horizontal location, $F=0.29$, $p=0.99$) (see Figure 46). However, when analysis is limited to either the vertical or horizontal location of the sensor, a significant interaction between group x vertical location is noted ($F=2.74$, $p=0.009$), but not for group x horizontal location ($F=1.85$, $p=0.84$) (see Figure 46). Post-hoc analysis of estimated marginal means according to group indicates that the proximal two channels in the injured group are relatively less active than those in the control group. This indicates a greater contribution to overall activity from the distal region of the previously injured hamstring muscle (see Figure 47). Analysis of rank transformed data according to the factorial analysis being considered confirms this finding. No significant differences between groups are noted using a three-way analysis (group x vertical location x horizontal location, $F=0.29$, $p=0.99$). However, a significant difference is confirmed between groups when a two-way analysis of group x vertical location is considered ($F=3.2$, $p=0.003$) while no difference is noted for the group x horizontal location interaction ($F=2.03$, $p=0.11$).

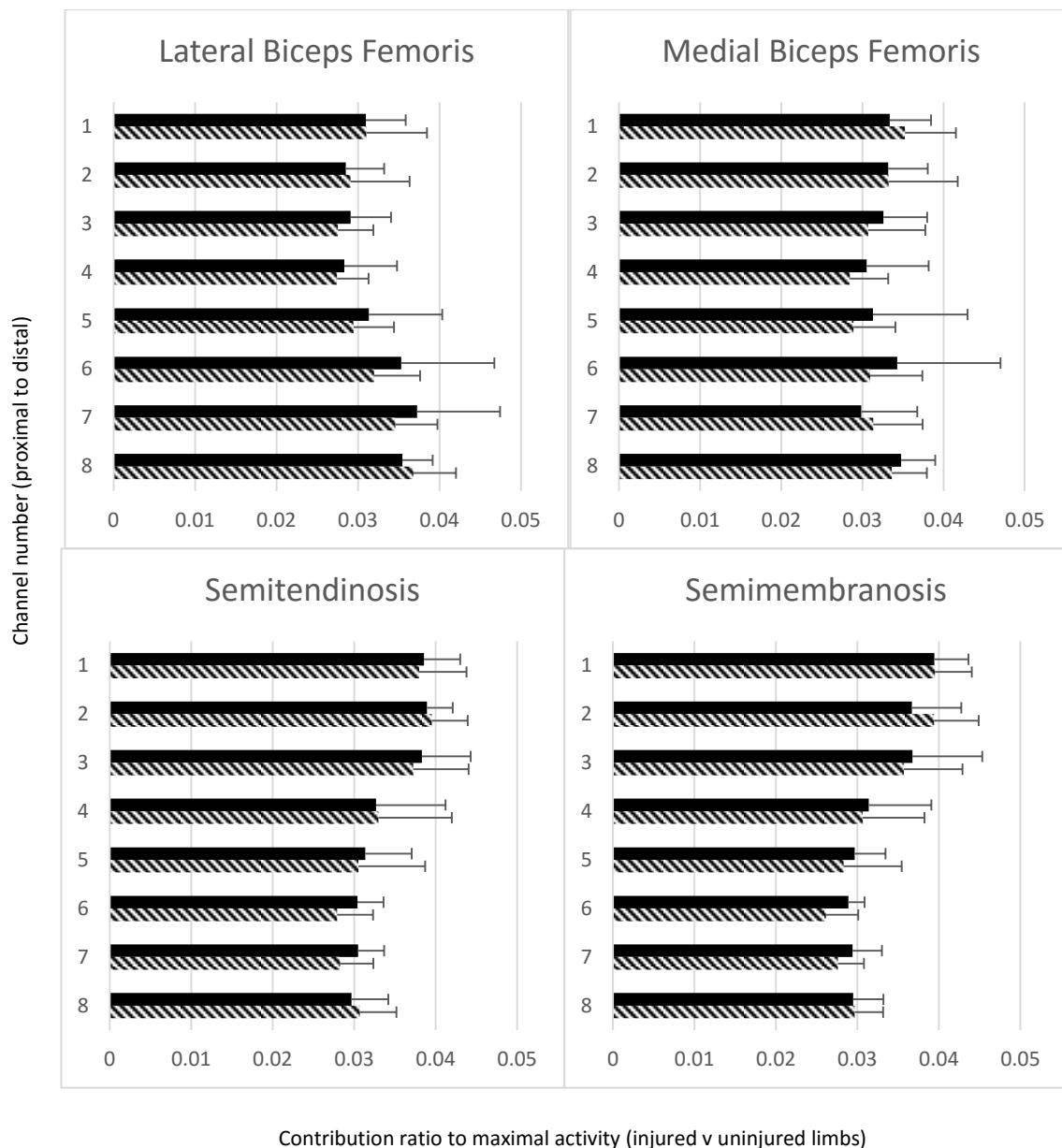


Figure 45: Relative electromyographic amplitude during the period of maximal electromyographic activity across the limb (32 channels). Channel numbers are aligned vertically (proximal to distal). Subplots indicate values for each of the horizontal locations nominally indicated by corresponding muscle (medial to lateral). Black bar= previously injured limb; Hatched bar = uninjured limb (in previously injured group). Error bar = one standard deviation.

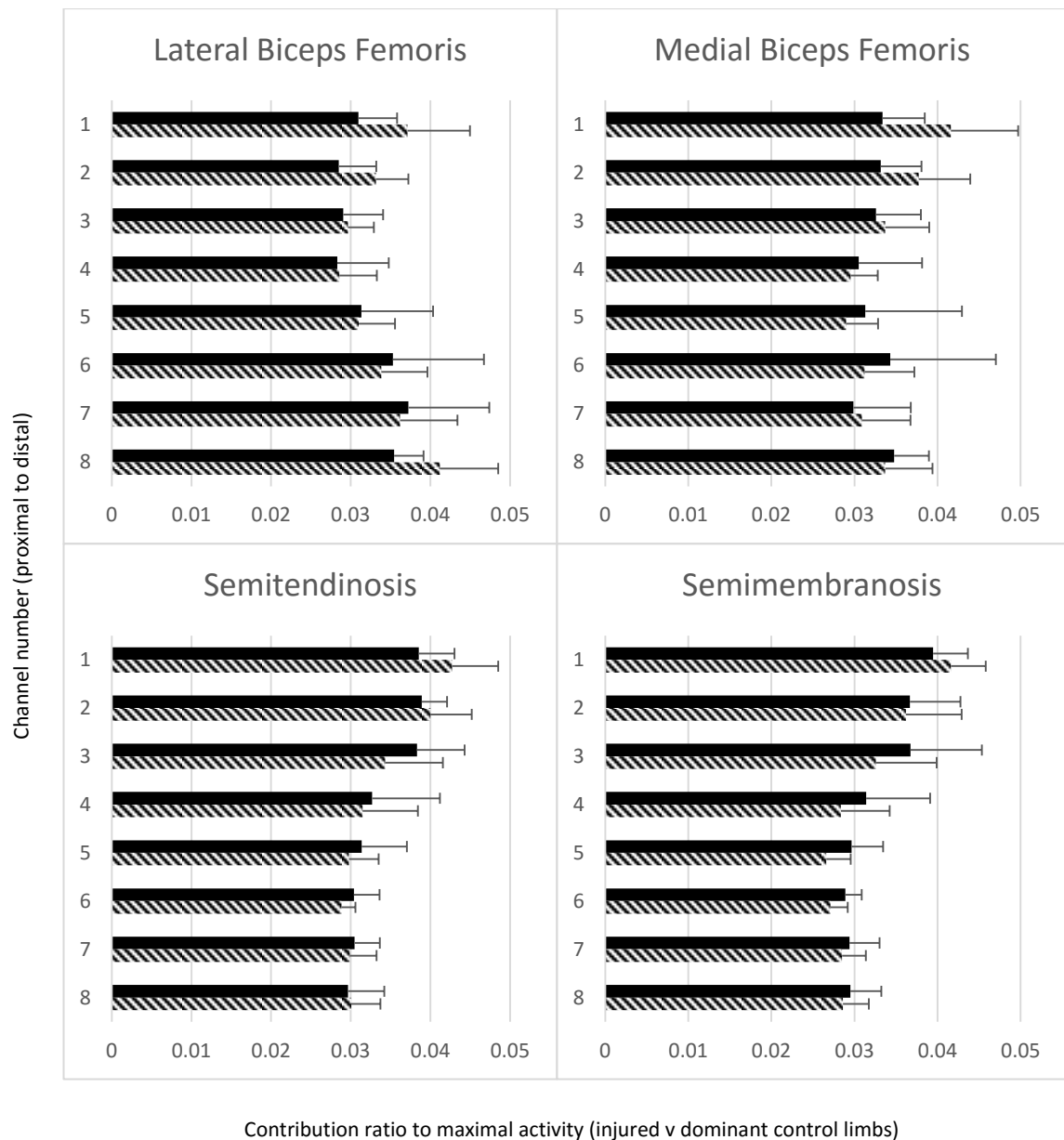


Figure 46: Relative electromyographic amplitude during the period of maximal electromyographic activity across the limb (32 channels). Channel numbers are aligned vertically (proximal to distal). Subplots indicate values for each of the horizontal positions nominally indicated by corresponding muscle. Black bar= previously injured limb; Hatched bar = dominant limb (in control group). Error bar = one standard deviation.

Estimated marginal mean of electromyographic contribution ratio

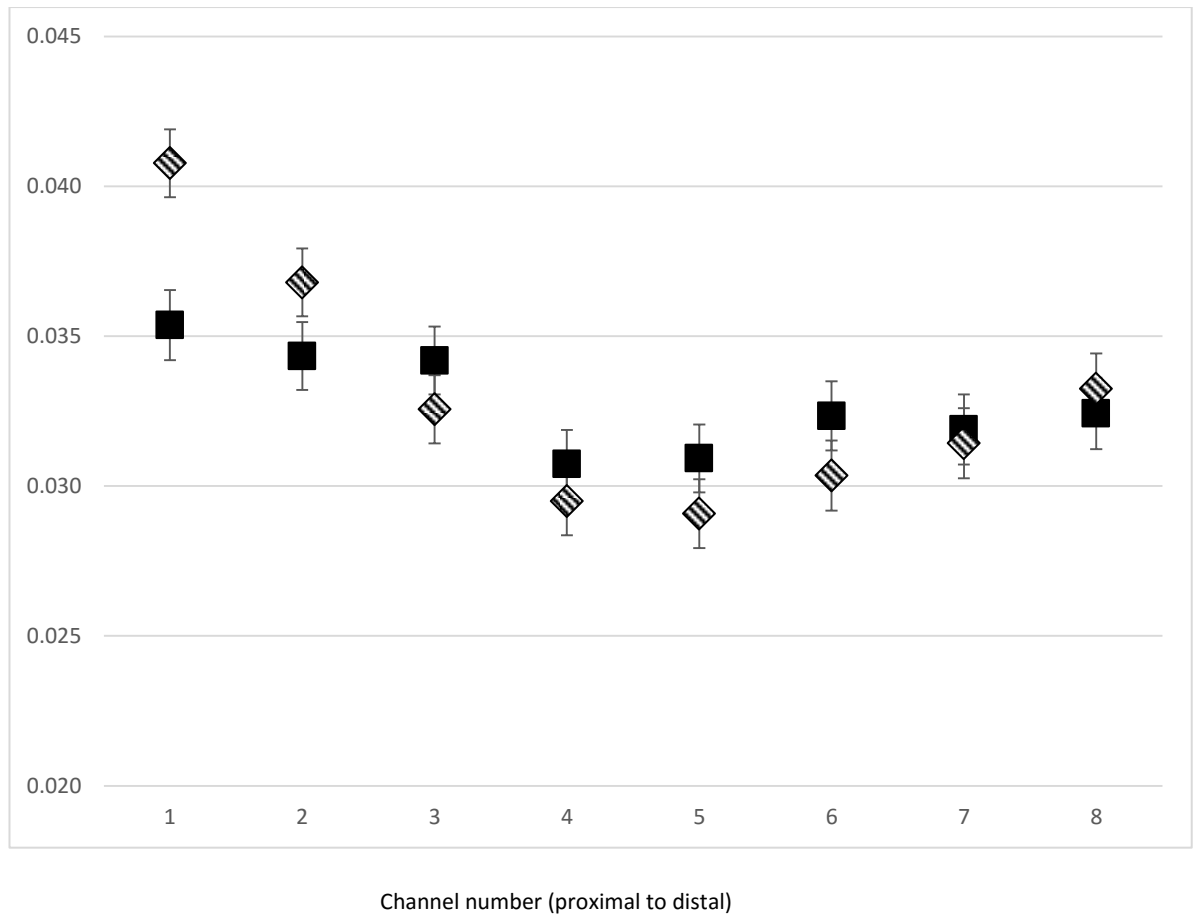


Figure 47: Estimated marginal means of the contribution ratio of each channel to overall maximal electromyographic activity according to longitudinal location of electromyographic sensor across all hamstring muscles. Data is subdivided according to group (Black square = previously injured limb; hatched diamond = dominant limb of control group). Error bars = +/- 1 standard error.

6.3.4.1 Effect size analysis

When this outcome is examined using effect size calculations, the largest effects are noted at the two most proximal channels (see Table 10).

Channel number	Cohens d
1	4.8
2	2.2
3	-1.4
4	-1.1
5	-1.6
6	-1.7
7	-0.4
8	0.7

Table 10: Effect size calculations for the group estimated marginal mean values for the maximal contribution data.

6.3.5 iEMG data

There was no significant difference in iEMG values between previously injured and uninjured limbs in the previously injured group for the full three-way factorial analysis (limb x vertical position x horizontal position $F=0.03$, $p=1.00$) or either two way analysis (limb x vertical position $F=0.15$, $p=0.99$; limb x horizontal position $F=0.11$, $p=0.95$). When comparison is made between previously injured limbs and the dominant limbs of the control group, no significant differences are noted in iEMG values for the three way analysis (group x vertical position x horizontal position, $F=0.05$, $p=1.00$) nor either two way analysis (group x vertical position, $F=0.03$, $p=1.00$; group x horizontal position $F=0.36$, $p=0.78$). Secondary analysis using data rescaled according to rank for each factorial interaction confirms that there is no significant difference between previously injured and uninjured limbs (three way limb x vertical location x horizontal location, $F=0.03$, $p=1.00$; two way limb x vertical location, $F=0.15$, $p=0.99$; two way limb x horizontal location $F=0.11$, $p=0.96$) and versus the dominant control limb (three way group x vertical location x horizontal location, $F=0.05$, $p=1.00$; two way group x vertical location, $F=0.03$, $p=1.00$; two way group x horizontal location $F=0.35$, $p=0.79$).

6.3.6 Frequency analysis

No significant differences for median frequency values exist between previously injured and uninjured limbs of previously injured athletes. This was true for the three way ANOVA

considering horizontal and vertical position of the sensors ($F=0.13$, $p=1.00$), as well as both two way ANOVA interactions (group x vertical position, $F=0.92$, $p=0.49$; group x horizontal position, $F=0.32$, $p=0.81$) (see Figure 48). Furthermore, there were no significant differences between the previously injured limb and the dominant limb of control subjects for the full three way analysis (group x vertical position x horizontal position, $F=0.43$, $p=0.99$), or the group x vertical two way analysis ($F=0.9$, $p=0.51$). However, a significant difference was noted for group x horizontal position of the sensor ($F=5.99$, $p=0.001$) (see Figure 49). Analysis of estimated marginal means indicates that the previously injured medial hamstring channels (semitendinosus and semimenranosis) display reduced median frequency values when compared to the dominant limb of the control group (see Figure 50).

Secondary analysis of the data following rescaling and rank alignment confirms that a significant difference in median frequency values exists between the previously injured limb and the dominant limbs of the control group (group x horizontal position $F=6.76$, $p<0.0001$). No significant differences are confirmed between the injured limb and uninjured limb of the previously injured group for the three-way factorial analysis (limb x vertical position x horizontal position, $F=0.13$, $p=1.00$) nor either two way analysis (group x vertical position, $F=1.02$, $p=0.41$; group x horizontal position, $F=0.42$, $p=0.74$). Furthermore, there are no significant differences are confirmed when comparison is made between the previously injured limb and the dominant limb of the control group for both the three way analysis (group x vertical position x horizontal position, $F=0.43$, $p=0.99$) and the remaining two way analysis (group x vertical position, $F=1.00$, $p=0.42$).

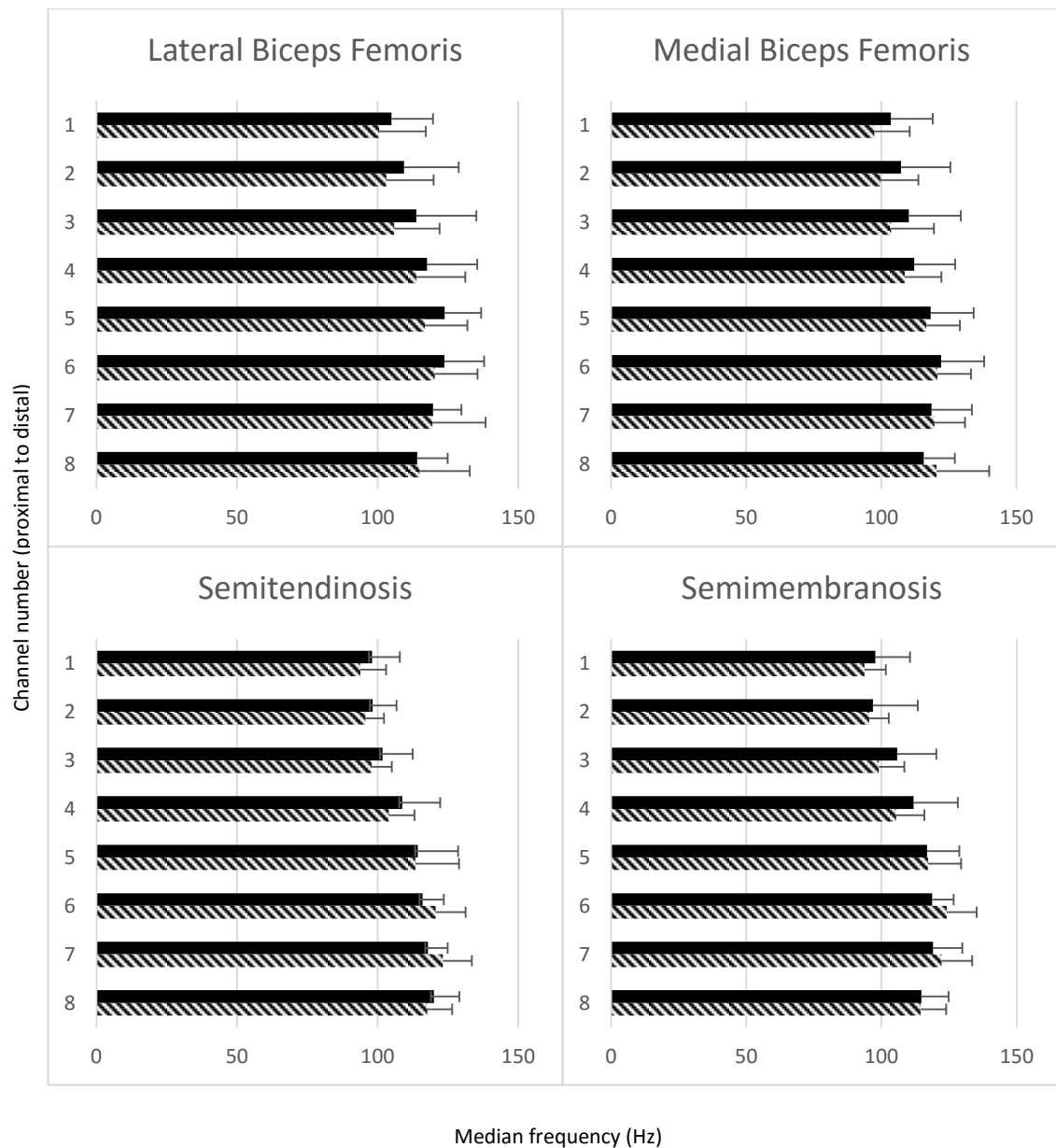


Figure 48: Median electromyographic frequency during loading period of a Nordic loading exercise. Channel numbers are aligned vertically (proximal to distal). Subplots indicate values for each of the horizontal positions nominally indicated by corresponding muscle. Black bar= previously injured limb; Hatched bar = uninjured limb (in previously injured group). Error bar = one standard deviation.

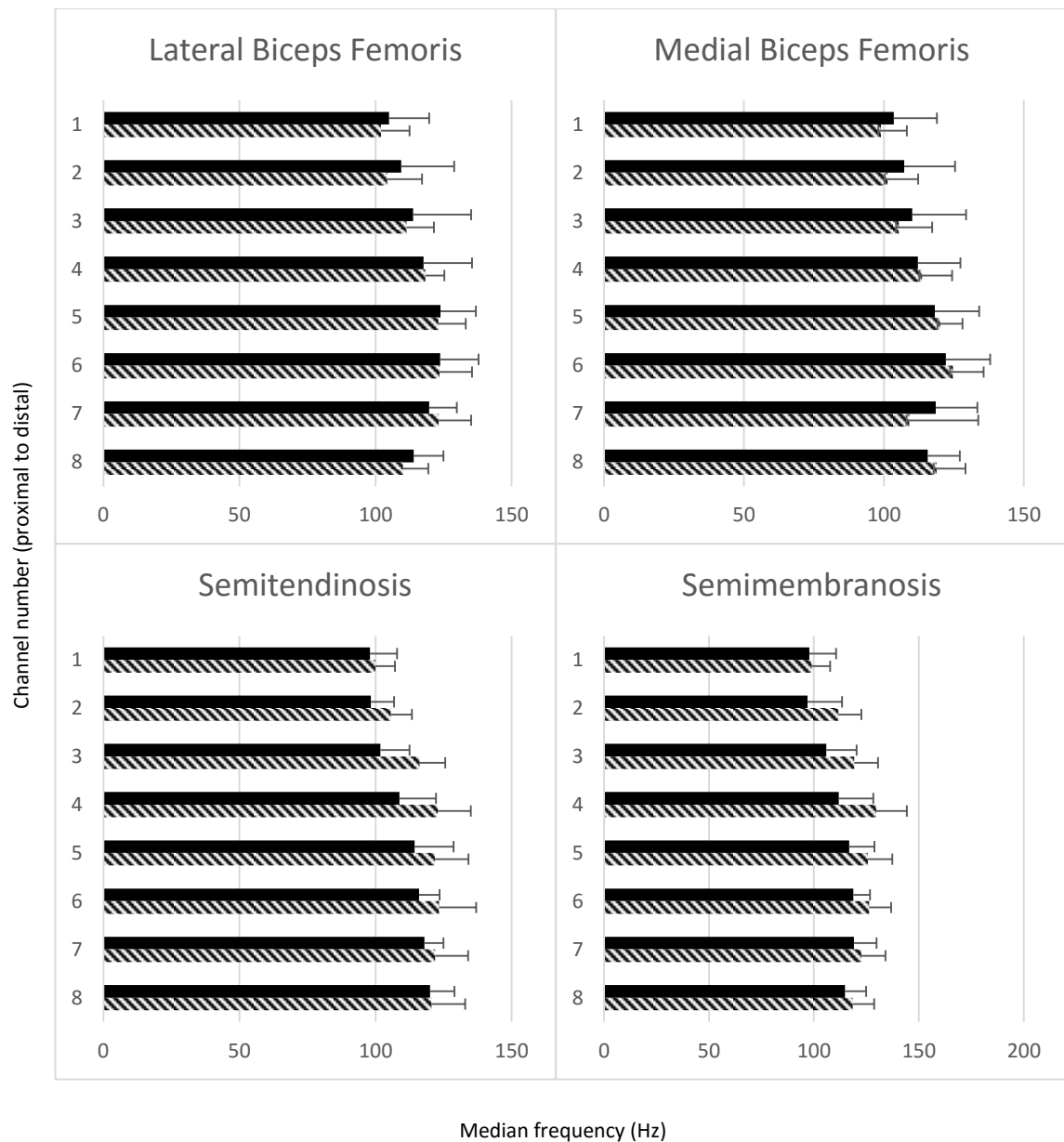


Figure 49: Median electromyographic frequency during loading period of a Nordic loading exercise. Channel numbers are aligned vertically (proximal to distal). Subplots indicate values for each of the horizontal positions nominally indicated by corresponding muscle. Black bar= previously injured limb; Hatched bar = dominant limb (in control group). Error bar = one standard deviation.

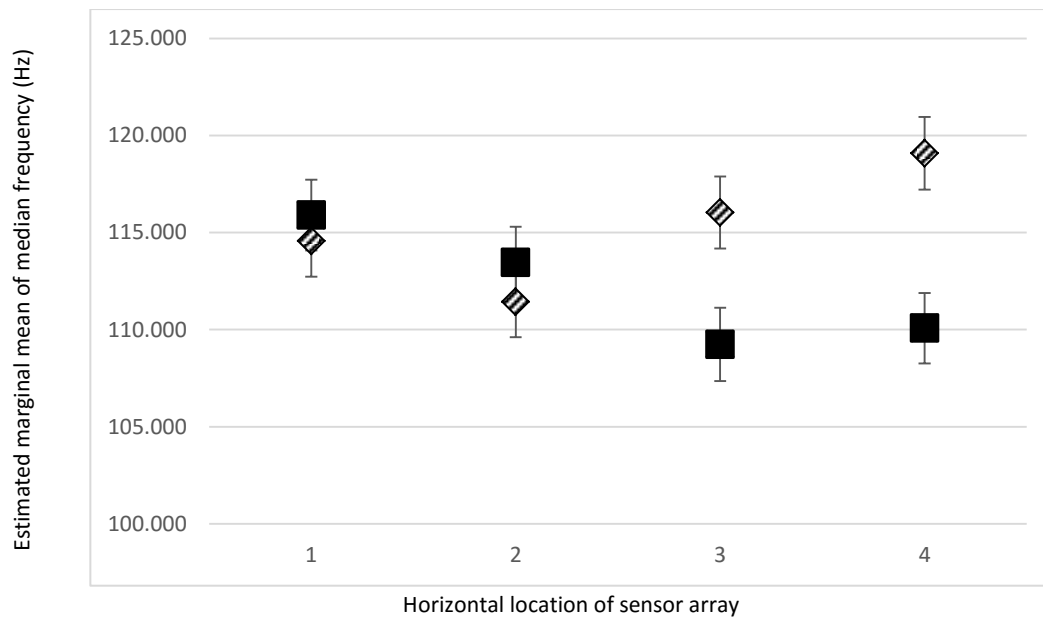


Figure 50: Estimated marginal means of the median frequency during loading according to horizontal location of the electromyographic sensor arrays. The numbers along the x axis correspond to the horizontal position of the sensor nominally corresponding to the underlying muscle as follows: 1=lateral biceps femoris, 2=medial biceps femoris, 3= semitendinosus, 4=semimembranosus. Data is subdivided according to group (Black square = previously injured limb; hatched diamond = dominant limb of control group). Error bars = +/- 1 standard error.

6.3.6.1 Effect size analysis

The effect size calculations based on the estimated marginal means for this outcome are presented in Table 11. This indicate large effects for the arrays overlying the medial hamstrings.

Array	Cohens d
1	-0.3
2	-0.4
3	1.3
4	1.8

Table 11: Effect size calculations based on the group estimated marginal means of the median frequency during loading according to horizontal location of the electromyographic sensor arrays. The array numbers correspond to the horizontal position of the sensor nominally corresponding to the underlying muscle as follows: 1=lateral biceps femoris, 2=medial biceps femoris, 3= semitendinosus, 4=semimembranosus

6.3.7 Comparison of limbs in control group.

We elected to make comparison between the previously injured limb and the dominant limb of the control group. In order to ensure that dominance was not unduly effecting the outcomes measured, a comparison of control limbs was conducted for all outcomes. No significant differences ($p>0.05$) were detected between limbs for the normalised EMG,

contribution ratio, iEMG using three-way ANOVA (group x vertical location of sensor x horizontal location of sensor) or either two-way analysis (group x vertical position of sensor; group x horizontal position of sensor. This was true for both the original data and that which was rank transformed and realigned according to the factorial interaction under consideration.

- Reduced proximal contribution to overall EMG signal in the injured hamstring compared to the dominant limb of the control group.
- Reduced median frequency in the previously injured medial hamstring muscles (semitendinosus and semimembranosus) compared to dominant limb of the control group.

Table 12: Summary of significant findings.

6.4 Discussion

This study is the first, to our knowledge, to present a detailed analysis of post injury hamstring activation patterns using low density, high surface area EMG. Significant reductions in contribution to overall maximal EMG activity were detected in the proximal region of the hamstring in previously injured subjects. Furthermore, frequency analysis indicates reduced median frequency values in the medial hamstring muscles of previously injured subjects. These findings were only apparent when comparisons were made to uninjured control subjects (dominant limbs). No differences were detected between the previously injured limb and uninjured limb of previously injured subjects. The control limbs were not statistically different from each other. Furthermore, no deficits in force generation capacity were detected between limbs in the previously injured subjects. This data suggests that previously injured athletes display altered EMG activity following return to sport post hamstring injury without displaying impairments in the ability to generate force.

Sprint related hamstring injury is predominantly limited to the long head of the biceps femoris (48, 54). Ekstrand *et al* (189) recently reported the location distribution of hamstring injuries at the elite levels across 46 European football clubs based on MRI scans. The majority of injuries (86% of 255 injuries) were confined to the mid-distal biceps femoris. A limitation of our work is that we did not obtain imaging to confirm the site of injury. Nevertheless, we detected that previously injured athletes display reduced

contribution from the proximal region of the hamstring to overall maximal activity suggesting that the injury associated changes in activity levels during the Nordic exercise may be remote to the actual injury site.

The Nordic loading exercise has been shown to preferentially load the semitendinosus with fMRI relation times noted to be 16.8% longer than the biceps femoris muscle in uninjured participants following loading (68). These authors also report a general reduction in activation levels in the previously injured muscle. The data reported in this study suggests that the median frequency values are reduced in the medial hamstring muscles of the previously injured limb. With the Nordic preferentially recruiting the medial hamstring, perhaps this result indicates more global inhibition on the injured side. Indeed a reduction in median frequency may occur secondary to a lack of recruitment of fast twitch fibres and/or larger motor units (130).

There is limited prior research investigated the use of low density, high surface area EMG sensor arrays on the hamstrings with one full study and two abstracts indicating that the hamstrings appear to activate in a consistent pattern during running, walking and isometric contractions using 16 channel arrays and a 20 channel linear array respectively (184-186). Other studies have shown alterations in spatial activation patterns in the quadriceps in response to delayed onset muscle soreness (161) and in the elbow flexors in response to fatigue (161). The consistent nature of spatial activation in the hamstring and the sensitivity of low density, high surface area EMG in detecting motor recruitment alterations provides a basis in using this application as a screening tool. Should a causal relationship be established between altered activation patterns and future injury, this approach may enable detailed assessment of post-injury spatial activation patterns in order to more accurately track recovery.

Our study groups had no interlimb or intergroup differences in their ability to generate force. This, in combination with the alterations in EMG activity patterns, suggests that the previously injured subjects may have developed compensatory strategies to regain their force generating capacity. Similar strategies are observed in gait following stroke and spinal cord injury and indicate a plasticity or adaptability within the motor control system (190-192). Perhaps the findings of this study are manifestations of such compensatory

change with a shifting of motor unit pool recruitment away from previously injured motor units in order to maintain function.

The study population used in this research was quite heterogeneous in terms of sporting level. This somewhat enhances the generalisability of these findings. The variance of this population in terms of sporting level and type indicate that these findings exist across a wide sporting population. Indeed, these findings were apparent with analysis of variance testing. This method does not fully respect the location of the sensor on the muscle and therefore intergroup differences must be reasonably large for detection of significant difference.

There is a lack of research using low density, high surface area EMG to measure hamstring muscle activity and there is no other study that we are aware of that measures spatial activation following hamstring injury. We therefore lack information about normal variance of activation in a larger population. However, we ensured that our control group was closely matched to the previously injured group. In this way we attempted to minimise the effects of confounding factors such as increased training exposure, increased body weight and general strength levels.

6.5 Conclusions

This study is the first to explore spatial hamstring muscle activation following return to sport following injury. We detected alterations in myoelectric characteristics in the previously injured group when comparison is made to controls during the Nordic loading exercise. Previously injured athletes display reduced contribution to maximal hamstring EMG activity in the proximal muscle region on the previously injured limb. Furthermore, significant reductions in median frequency values were detected on the medial aspect of the hamstring group of the previously injured subjects. Force measures were symmetrical between group and limbs. The measurement of spatial hamstring activation patterns using low density, high surface area EMG may offer valuable potential in tracking recovery following hamstring injury.

7 Case Studies

Hamstring muscle function during injury rehabilitation – two case studies

7.1 Introduction

Hamstring injuries are common in sports which involve sprinting. Across many sports hamstring injuries are the most common injury encountered accounting for between 6 and 36% of all injuries recorded (2-4, 9, 11, 67). Indeed hamstring injury appears to be becoming more common with a 4% annual increase in incidence measured since 2001 in elite European football (14). Despite changes in diagnostic and rehabilitation approaches, re-injury rates have remained persistently high. Indeed, previous hamstring injury is the lead risk factor for future injury, accounting for up to an 11-fold increase in risk over a subsequent season (2, 11, 14, 20). Current rehabilitation approaches include sport specific loading programmes (60, 63), a focus on high intensity eccentric loading (64) and postural control and agility work (62). Specifically, the addition of high intensity slow eccentric loading in the form of a Nordic loading protocol, seems to reduce re-injury rates in compliant athletes (64). However, the propensity for the hamstring re-injury remains a major issue.

Recently, the theory of persistent post-injury neuro-inhibition has been proposed as a mechanism underlying elevated re-injury risk. Fyfe et al (61) propose that poor rehabilitation practices may lead to painful inhibition of muscle activity, the development of excessive scarring, shorter optimal muscle lengths and muscle atrophy. Evidence to support this theory are based on known risk factors for hamstring re-injury, in particular altered strength profiles (11, 20, 32). There is also some limited evidence of association between prior injury and persistently altered muscle activity in the form of bipolar s-EMG and fMRI findings. Specifically, previously injured athletes appear to display a reduced ability to recruit their previously injured biceps femoris following return to sport (68-70, 103).

Standard bipolar EMG provides general information relating to the motor unit action potentials generated within range of the sensors (130, 158). In larger muscle groups, such as the hamstring, large areas of the muscle remain outside the detection area of the electrode. It is known that EMG activity appears to be non-uniform across muscle bellies

with different regions within a muscle contribute in differing ways during a contraction (158-162). Several studies have explored the regional distribution of EMG activity using low density, high surface area arrays of sensors with outcomes related to fatigue and delayed-onset muscle soreness predominating (159-163). Despite extensive searching of the literature, no studies were identified which explored spatial activity in the early stages following an acute hamstring injury.

The aim of this project was to explore early post-injury hamstring function in two elite athletes in the early stages following a biceps femoris injury. Measures of force production capacity were made using an isokinetic dynamometer in one athlete and a strain gauge in the other. Simultaneous analysis of EMG activity across the hamstrings during force production were made using linear arrays of monopolar EMG sensors aligned longitudinally over the hamstring muscles. The alternative hypothesis was that that there would or would not be detectable asymmetries in activation patterns between previously injured and uninjured limbs at the time of return to sport. The null hypothesis was that no asymmetries would be present.

7.2 Case 1

7.2.1 *Methods*

A professional female sprinter (Age 19 years; weight 55kg; height 1.65m) was assessed on one occasion in the Queen Mary University of London human performance laboratory. Ethical approval for the study was provided via university ethics review and the athlete gave written consent for their data to be used within this report.

7.2.1.1 *Athlete profile:*

This athlete competes at international level and has medalled at various world championships. Subsequent to this testing, she medalled at international competition.

7.2.1.2 *Injury profile (based on verbal medical report):*

Prior to testing this athlete has sustained a series of right biceps femoris injuries in the previous two years. The most recent injury had occurred 5 weeks prior to the testing session during a sprint training session. The athlete described a sudden onset, proximal hamstring pain which occurred during a sprint. Follow-up MRI confirmed a Grade 1 injury to the proximal myotendinous junction of the biceps femoris. The athlete had undergone

progressive rehabilitation with her physiotherapist which included running training of increasing duration and speed, progressive eccentric focussed hamstring loading alongside multi-joint lower limb loading and postural control work closely matching the rehabilitation protocols outlined in published research (60, 62, 63). This athlete did sustain another Grade 1 right hamstring injury within 12 months of testing. However, she subsequently recovered and continues to compete at the highest levels.

7.2.1.3 Data collection protocol

7.2.1.3.1 Isokinetics

An isokinetic profile of knee flexion and extension strength was obtained using an Isocom Dynamometer (Eurokinetics Ltd, UK). The athlete was seated in an upright position with approximately a right angle at the hip. The athlete's thigh was firmly secured to the seat using a harness system. The athlete's trunk was further supported by a four-point seatbelt. Further stability was enabled by instructing the athlete to hold the handles on either side of the seat. Once in position and secured, the fulcrum of the dynamometer was positioned to align with the transverse axis of the knee joint. The athlete's lower leg was secured to the arm of the dynamometer using heavy duty straps (see Figure 51). Finally, the range of motion limits were set for knee flexion and extension and the subject was provided with an opportunity for familiarisation through a series of low intensity test trials. The dynamometer was calibrated according to manufacturer recommendations prior to testing.



Figure 51: Isokinetic dynamometer setup for knee flexion and extension testing (taken from isokinetic.com)

The testing session consisted of nine trials for each limb incorporating five familiarisation trials and four test trials. Torque data was collected at 200hz during each trial. Thirty

second reset periods were given between trials. The sequence of testing is outlined in Table 13. The athlete was given loud verbal encourage to perform maximal effect contractions against the resistance of the dynamometer for each test trial. Data was collected from the previously injured limb (right) followed by the left with the athlete performing three repetitions for each trial.

	Knee Flexor Effort			Knee Extensor Effort		
	Mode	Velocity (°/second)	Repetitions	Mode	Velocity (°/second)	Repetitions
Trial 1-3	Con	60	3	Con	60	3
Trial 4-5	Con	240	3	Con	240	3
Trial 6-7	Ecc	30	3	Ecc	30	3
Trial 8-9	Ecc	120	3	Ecc	120	3

Table 13: Data collection procedure for isokinetic dynamometry (Con=concentric; Ecc=eccentric)

The contraction velocities outlined in Table 13 were chosen in agreement with previously published data reported post hamstring injury isokinetic dynamometry (112, 113, 117, 118)

7.2.1.3.2 Electromyography

Surface EMG signals were collected from the posterior thigh region using four, eight channel monopolar linear arrays. The athletes skin was prepared by shaving, cleaning with alcohol gel and lightly abrading the skin surface to maximise sensor adherence and reduce impedance. This approach is in agreement with international guidelines (136). The monopolar electrodes were placed at 20mm intervals with the central pair of electrodes located either side of a mark placed in the anatomical centre of the biceps femoris, and combined medial hamstrings with reference to SENIAM guidelines (136). Two linear arrays were placed either side of line drawn along the anatomical centre of the biceps femoris and medial hamstring muscles (Figure 52). The accuracy of the location of the arrays was confirmed using palpation by the researcher, an experienced musculoskeletal physiotherapist. In total, data was collected from 32 monopolar sensors at 2048hz for the duration of each trial.

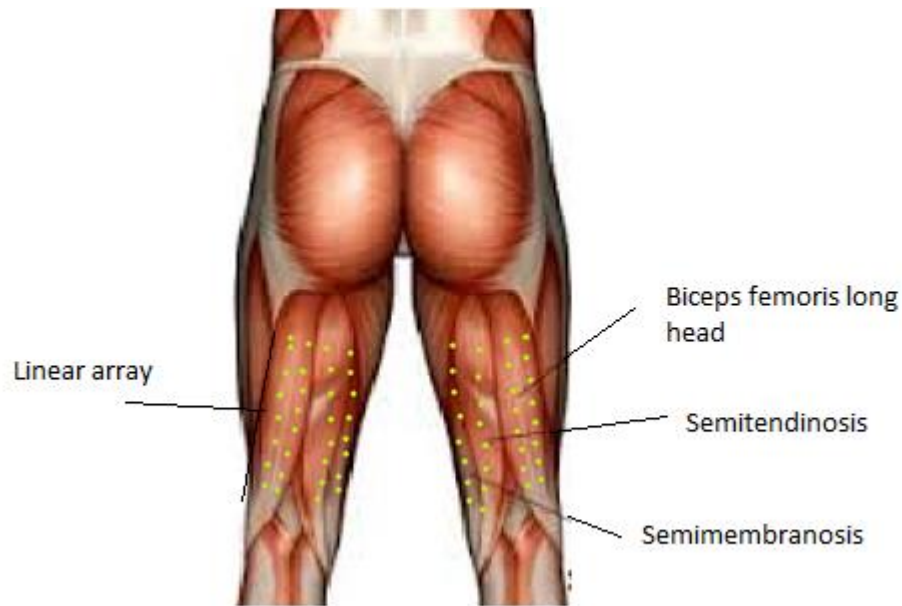


Figure 52: Location of electromyographic sensor (Green dots) according to the anatomical orientation of the hamstrings

7.2.1.4 Data analysis

Isokinetic data was analysed using the proprietary software. Peak torque, peak torque (normalised to body weight), angle of peak torque and total work were calculated for each test trial for each leg and muscle group tested. The peak torque measure was the single maximum values from across the three trials. Ratios of peak torque between the knee flexors and extensors were calculated separately.

EMG data was analysed offline using custom written Matlab programs (Version 2014b, Matworks Ltd, USA). Signals from all 32 channels were band pass filtered (10-500Hz) in line with international recommendations (136, 164). A notch filter at 50hz was applied to remove interference from ambient electrical noise. The filtered data was inspected for quality and any signal adverse effected by motion artefact was removed from further analysis.

The EMG analysis focussed on identifying the regional distribution of maximal activity in the previously injured and uninjured hamstrings. The period of maximal EMG activation during each isokinetic trial (all three repetitions together) was calculated by averaging rectified activity from all 32 channels together. All timepoints were identified where mean

EMG activity across the 32 channels on each limb was within 20% of maximum. This calculation was made on data that was smoothed using a 500 point (~25ms) running window to avoid undue influence from momentary high amplitude signal. The contribution of each channel to maximal activity across all channels was calculated using unsmoothed data and expressed as a ratio of contribution for each channel where maximal activity was considered as a value of 1. In this way, a spatial representation of maximal activity contribution was obtained for each available EMG channel.

7.2.1.5 Data reporting

Data is presented in descriptive form. Test of significance were not performed as they are not appropriate in case study design. Comparisons are made between the previously injured (right) and uninjured limb.

7.2.2 Results

7.2.2.1 Isokinetics

The results of the isokinetic tests related to hamstring function are presented in Table 14. Data related to knee extensor function is omitted with the exception of the hamstring to quadriceps peak torque (H:Q) ratio for each velocity. With the exception of the concentric 60°/second trial, peak torque values are elevated on the previously injured side. This athlete was noted to be particularly strong on eccentric effort with a torque/body weight percentage of 285.1% noted during the 120°/second trial on the previously injured limb. The ratio of hamstring peak torque to quadriceps peak torque was noted to be generally lower across both limbs during concentric contractions compared to eccentric contractions. The angle of peak torque was broadly similar between limbs with the exception of the 60°/second concentric trial.

7.2.2.2 Electromyography

The analysis of the channel by channel contribution to maximal EMG activity indicates notable asymmetries in activation patterns across all contraction types and velocities. Data relating to biceps femoris activity is presented in Figure 53. There appears to be increased contribution to maximal hamstring activity in the region of the proximal biceps femoris of the injured limb when compared to the uninjured side.

Knee flexors (60°/second - concentric)		
	Left	Right*
Peak torque (Nm)	92.8	69.9
Peak torque/BW (%)	165.8	124.8
Angle of peak torque (°)	18.9	51.5
Total work (J)	226.8	171.6
H:Q ratio	0.52	0.37
Knee flexors (240°/second - concentric)		
	Left	Right*
Peak torque (Nm)	59.3	64.4
Peak torque/BW (%)	105.9	114.9
Angle of peak torque (°)	21.8	20.8
Total work (J)	166.5	181.2
H:Q ratio	0.58	0.51
Knee flexors (30°/second - eccentric)		
	Left	Right*
Peak torque (Nm)	130.8	144.3
Peak torque/BW (%)	233.5	257.6
Angle of peak torque (°)	45.2	40.4
Total work (J)	255.6	329.4
H:Q ratio	1.69	1.38
Knee flexors (120°/second - eccentric)		
	Left	Right*
Peak torque (Nm)	93.6	159.6
Peak torque/BW (%)	167.1	285.1
Angle of peak torque (°)	30.4	48.1
Total work (J)	113.7	451.9
H:Q ratio	1.43	1.57
Mixed peak torque ratio (Eccentric hamstring 30°/second: Concentric Quadriceps 240°/second)		
	Left	Right*
Mixed (functional) H:Q	1.29	1.14

Table 14: Isokinetic results. The asterisk (*) denotes the previously injured limb (BW= body weight).

This is true across both concentric and eccentric trials. Data relating to medial hamstring contributions are presented in Figure 54. In contrast to the biceps femoris, the proximal medial hamstrings of the previously injured limb appear to contribute less to maximal activity across all tests when comparison is made to the uninjured limb.

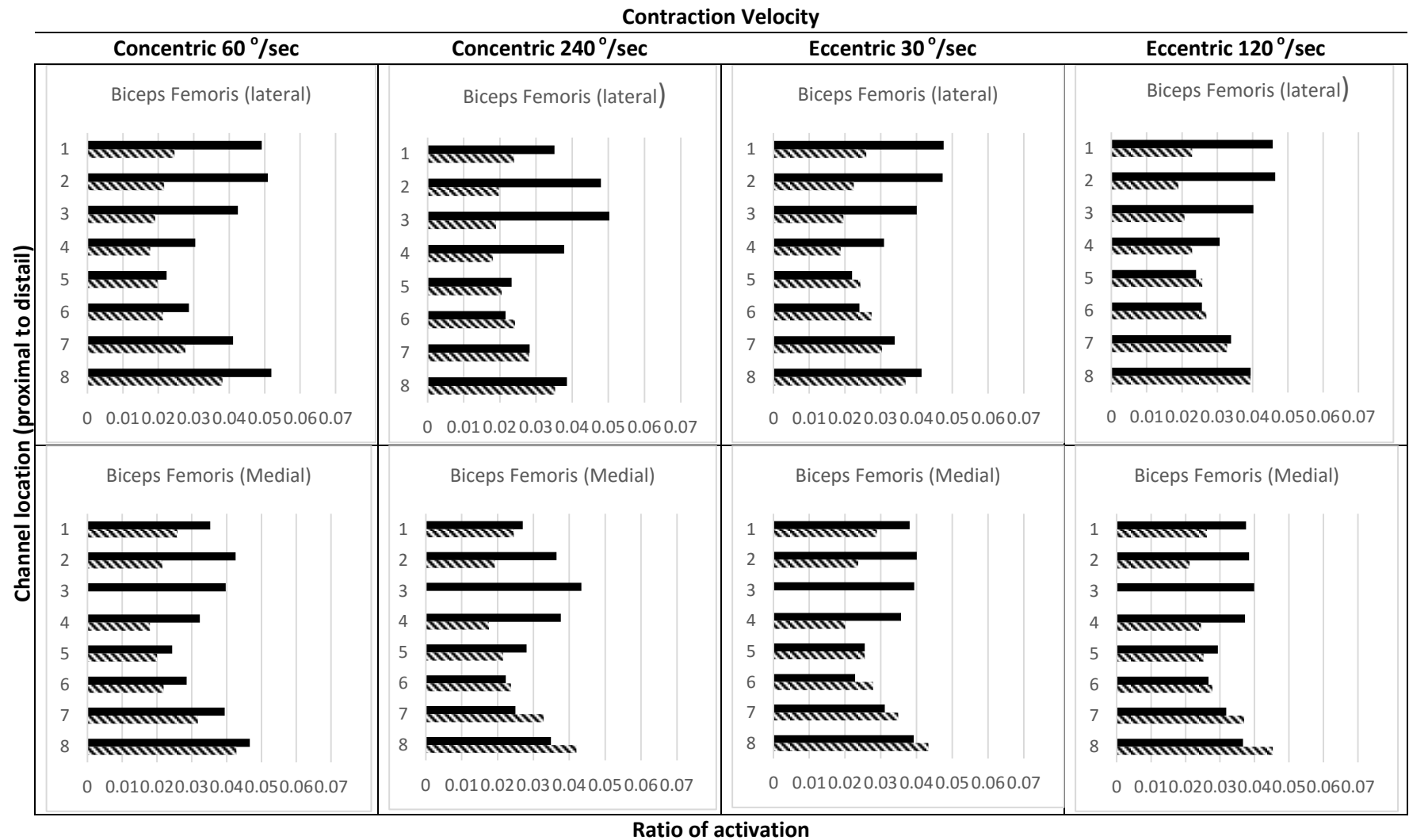


Figure 53: Channel contribution of the biceps femoris (medial and lateral) to maximal overall hamstring activity during each of four isokinetic contractions. Total activation =1. Previously injured limb = black. Uninjured limb = hatched

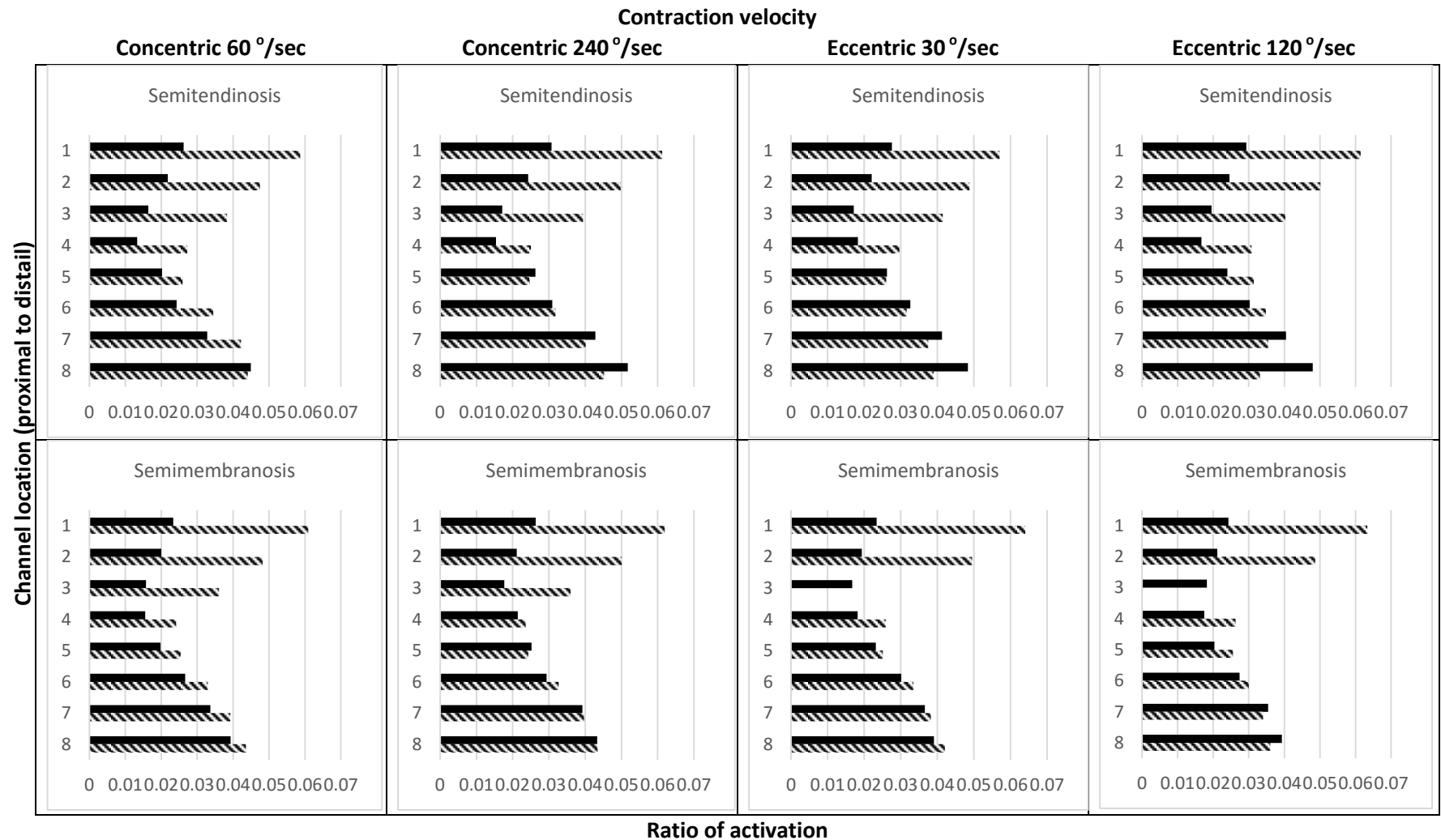


Figure 54: Channel contribution of the medial hamstrings to maximal overall hamstring activity during each of four isokinetic contractions. Total activation =1. The arrays of electrodes are nominally referred to as semitendinosus for the more lateral array and semimembranosus for more medial array. Previously injured limb = black. Uninjured limb = hatched

7.3 Case 2

A professional male footballer (age 19, weight 79kg, height 1.86m, playing position Centre Midfield) attended the Queen Mary University of London on three separate occasions following a right sprint related hamstring injury. This athlete provided voluntary written consent for their data to be used in the report. Prior ethical approval for this research was obtained via university ethics committee. Measures of isometric knee flexor force capacity and spatial s-EMG activation across both hamstrings were collected as he progressed through rehabilitation and return to full participation in sport.

7.3.1 *Methods*

7.3.1.1 *Athlete profile*

At the time of assessment this footballer was signed to an English Premiership club and had several first team caps. He was a starting player on the club's under 21 team and had progressed through the club at all age levels over the previous 6-7 years. This footballer also had international caps at all levels up to Under 21.

7.3.1.2 *Injury and assessment profile*

This footballer sustained a right side hamstring injury during a pre-season match. He described sudden onset posterior thigh pain when sprinting for a ball. An MRI performed two days after the injury reports a grade 2 partial tear of the right long head of biceps femoris muscle at the musculotendinous junction within the middle third of the thigh (see Figure 55). This injury occurred three weeks following a grade 1 injury which was located adjacent to the current injury. This footballer had sustained a similar injury 17 months previously. The MRI report also notes some disruption of the intramuscular tendon of the biceps femoris. This footballer initially presented for assessment five weeks following the injury. Given the quick succession of injuries, the club medical team had elected to rehabilitate this player with extra caution. A careful progression of incremental loading had been initiated with an initial focus on isometric load followed by a slow introduction of eccentric load. Following the initial assessment in the laboratory, rehabilitation was progressed to include a progressive running programme as per Silder et al (63), more complex and dynamic loading exercises as per Heidersheit et al (60), and eventually a Nordic loading programme as per Petersen et al (64).

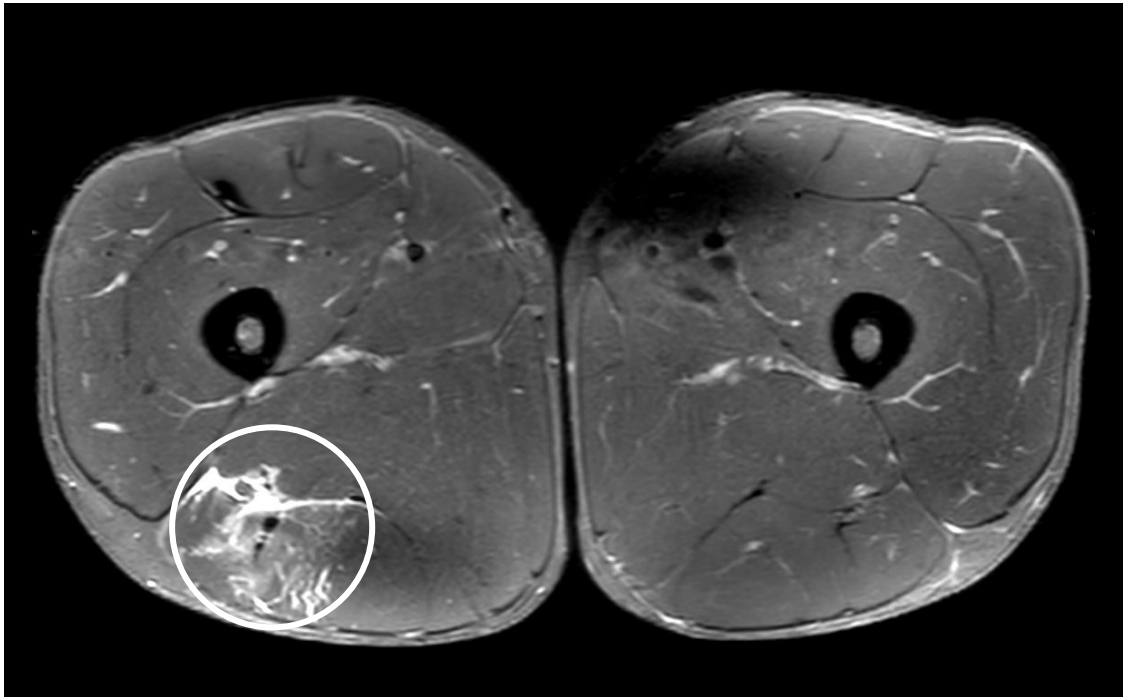


Figure 55: Magnetic resonance image showing an acute Grade 2 tear of the long head of the right biceps femoris. The area of the resultant haematoma is highlighted with the white circle. This image was taken two days post-injury

At eleven weeks post-injury, the footballer was undertaking some more intensive loading work (prone leg curl with eccentric focus) and felt some discomfort in the hamstring. A repeat MRI was conducted eleven weeks following the injury (and one week following the second testing session in this study) which indicated full resolution of the haematoma, mature scar tissue and thickening of biceps musculotendinous junction. Some minor oedema was noted in the semitendinosus which was presumed to be the origin of the discomfort (see Figure 56).

The footballer returned to full training activities 14 weeks after the initial injury. Unfortunately, two months later he sustained a grade 3 rupture of the same muscle. This required surgical intervention and resulted in an 18 month rehabilitation. The footballer was subsequently loaned out to a League 2 team where he re-injured his right hamstring after several games. His contract was not renewed for the following season and he remains out of contract (Aged 20).

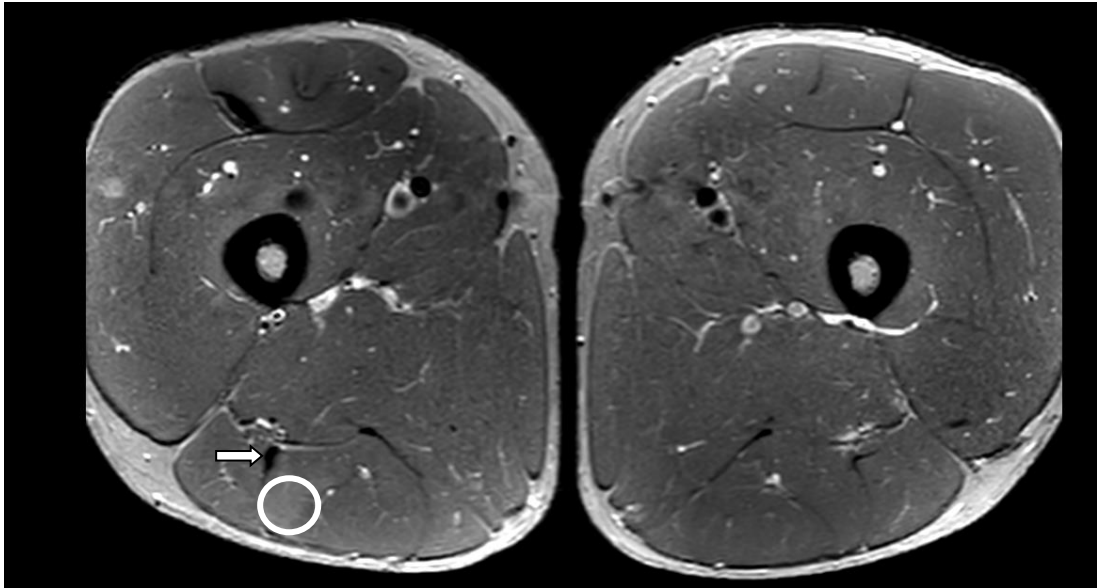


Figure 56: Magnetic resonance image of both thighs. Scar tissue in right biceps femoris at musculotendinous junction indicated with arrow. Mild oedema in semitendinosus indicated with circle. This image was taken eleven weeks post-injury.

7.3.1.3 Experimental procedure

The footballer underwent identical tests of hamstring muscle function during each of the three testing sessions. Testing sessions took place at five, eleven and fourteen weeks post-injury, the final occurring immediately prior to return to sport. In order to reduce the risk of re-injury during testing, a loading protocol was selected which enabled the subject to modulate effort based on any subjective perception of discomfort. As dynamometry and Nordic loading involve maximal effort, these methods were deemed high risk and therefore unsuitable in this case.

7.3.1.3.1 Measures of force

The footballer's ability to generate knee flexion force was determined through the use of a strain gauge ((TSA Alloy Steel S Type, Coventry Scales Company, UK). This was attached to the footballer's ankle via a heavy duty ankle cuff with the other end attached to an adjustable nylon strap, which in turn was firmly secured. Voltage output from the strain gauge was collected using a REFA64 device (TMSI Ltd, Netherlands) at 2048hz. The output was converted to kilogram weight using a conversion factor determined via separate calibration experiment. Peak isometric force was measured over three trials at three difference knee flexion ranges. An average across the three trials was calculated. The subject performed maximal effort contractions while lying in prone of a stable examination

bench. The first three contractions were performed with the knee positioned in full extension, the next three with the knee in 45° flexion and the final three with the knee at 90° flexion. The differing positions enabled separate analysis at differing functional ranges. During all trial the subject was given strong verbal encouragement to perform maximal effort contractions. At no stage did the subject complain of discomfort. Measurement were conducted on each limb independently starting with the uninjured side.

7.3.1.3.2 Electromyography

Simultaneous measures of s-EMG were collected at 2048hz using the same REFA64 system (TMSI Ltd, Netherlands). As with the other case in this chapter, and the Nordic loading protocol in Chapter 6, four, eight channel linear unipolar s-EMG arrays were attached the skin overlying the hamstring muscle. A ground electrode was positioned on the calcaneum. The skin was prepared by shaving, abrading and cleaning with alcohol gel as per SENIAM guidelines (136). The arrays were aligned to the anatomical location of the biceps femoris and medial hamstring muscles, with two arrays overlying each in a longitudinal orientation. The electrodes were positioned at 20mm intervals with the central two lying either side of the muscle midpoint measure according to SENIAM guidelines. The accuracy of positioning was confirmed via direct palpation in line with published recommendations (131).

7.3.1.4 Data analysis

All data was analysed offline using a custom written Matlab program. Force data was extracted for each trial, checked for quality and converted from voltage to kilogram weight using a known conversion factor. This was calculated in a separate experiment (see section 4.4.3.2). The EMG data was extracted and inspected for quality. Any missing channels were noted. The signals were band pass filtered (Ideal filter 10-500hz). A 50hz notch filter was also applied to remove ambient signal interference. Any remaining poor quality signals following filtering were removed from further analysis.

The aim was to explore the spatial contribution to maximal EMG activity across the entire hamstring. The period of maximum activation was identified by considering a summation of rectified amplitude of all channels on each leg. We smoothed the resulting signal by averaging across 500hz (~25ms) running windows in order to remove momentary high amplitude spikes. The maximum value of the smoothed average of signals was recorded. Using the non-smoothed rectified signal, we measured the contribution of each signal

during the periods when the average signal amplitude was within 20% of the peak amplitude. Each channel's specific contribution to maximal (>80%) amplitude was expressed as a ratio of total contribution, with maximal activation equalling 1. The location of each channel was preserved in order to give a spatial distribution of maximal EMG activity.

7.3.2 Results

7.3.2.1 Force production

This athlete was asked to contract his knee flexors while lying prone. Previously injured and uninjured limb peak force values at each knee flexion angle are presented for three testing sessions. In general peak force values were highest on both limbs when the knee was flexed to 45°. The lowest values were detected during the 90° flexion trials. This finding is unsurprising. The ability to generate force is optimal in the mid to inner range of skeletal muscle (193). The overlapping actin and myosin filaments sarcomere have most potential to shorten the muscle in this range. At the outer range, minimal cross bridges are possible due to a lack of overlap, while in the inner range, the ends of the filaments abut the z discs, thus preventing further contraction. At the first test session, there is clear asymmetry in peak force values between the weaker injured and uninjured limb at all positions. In general, this asymmetry reduced overtime but some weakness is seen to persist on the previously injured side at the final session particularly during the 0° and 45° contractions. It is noted that the footballer appeared weaker during the 45° knee flexion contraction in the second session. There is also reduced peak forces noted across all positions on the uninjured limb when the second session is compared to the first. These reduced force levels may be secondary to muscle soreness and semimembranosus oedema seen on MRI (see Figure 56). This appears to have been associated with an increased training intensity around that time perhaps having caused some delayed onset muscle soreness.

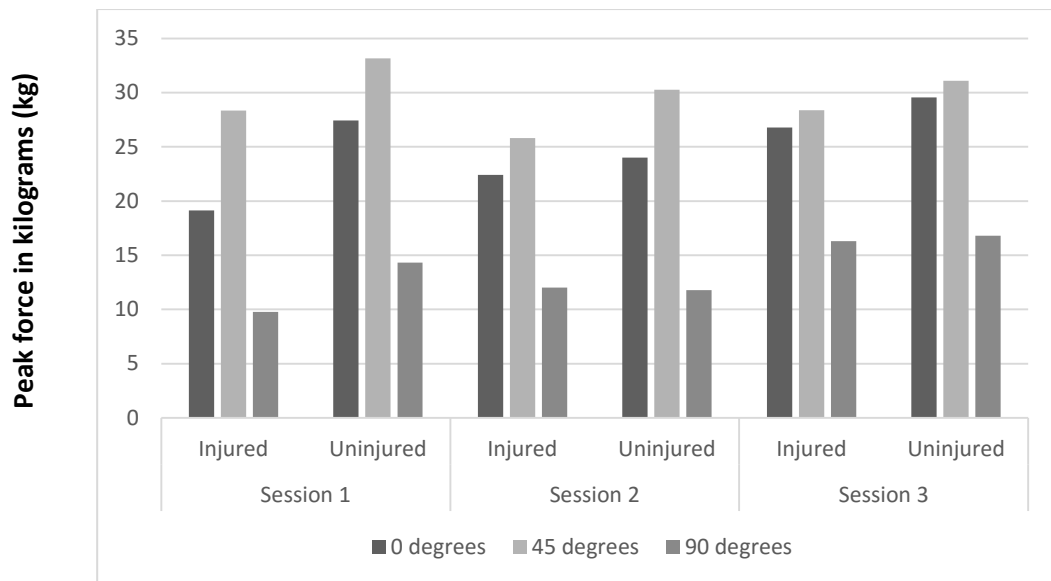


Figure 57: Maximum force produced during isometric contractions for three knee flexion positions over three testing sessions.

7.3.2.2 Electromyography

In the first testing session (five weeks post-injury), there appears to be a reduced contribution to overall maximal EMG activity in the region of the previously injured mid-distal biceps femoris (see Figure 58). Conversely, the mid-distal medial hamstrings on the previously injured side appear to contribute at increased levels compared to the uninjured limb. The reduced activity in the biceps femoris approximates to the area of prior injury based on the MRI report and image (Figure 55). These findings are most obvious in the mid (45 degrees) and outer (0 degrees) functional range contractions.

During the second testing session (eleven weeks post-injury) increases in contribution throughout the biceps femoris on the previously injured limb are noted when compared to the uninjured side (see Figure 60). There appears to be reduced levels of contribution in the proximal region of the medial hamstring in the previously injured limb. Again, these findings are most obvious during the mid and outer range contractions. These findings coincide with a period of increased eccentric loading and associated muscle soreness with semitendinosus oedema noted on MRI (Figure 56).

Finally, testing at 14 weeks post-injury reveals some persistent reduction in contribution to maximal activity in the region of the distal biceps during the mid and outer range

contraction (Figure 62). In contrast, the inner range contraction (0°) indicates elevated activation levels in the same region suggesting that the muscle activates differently at various functional length. The medial hamstrings show grossly symmetrical activation patterns in the mid and outer ranges and reduced contribution distally in the inner range contraction on the previously injured limb (Figure 63).

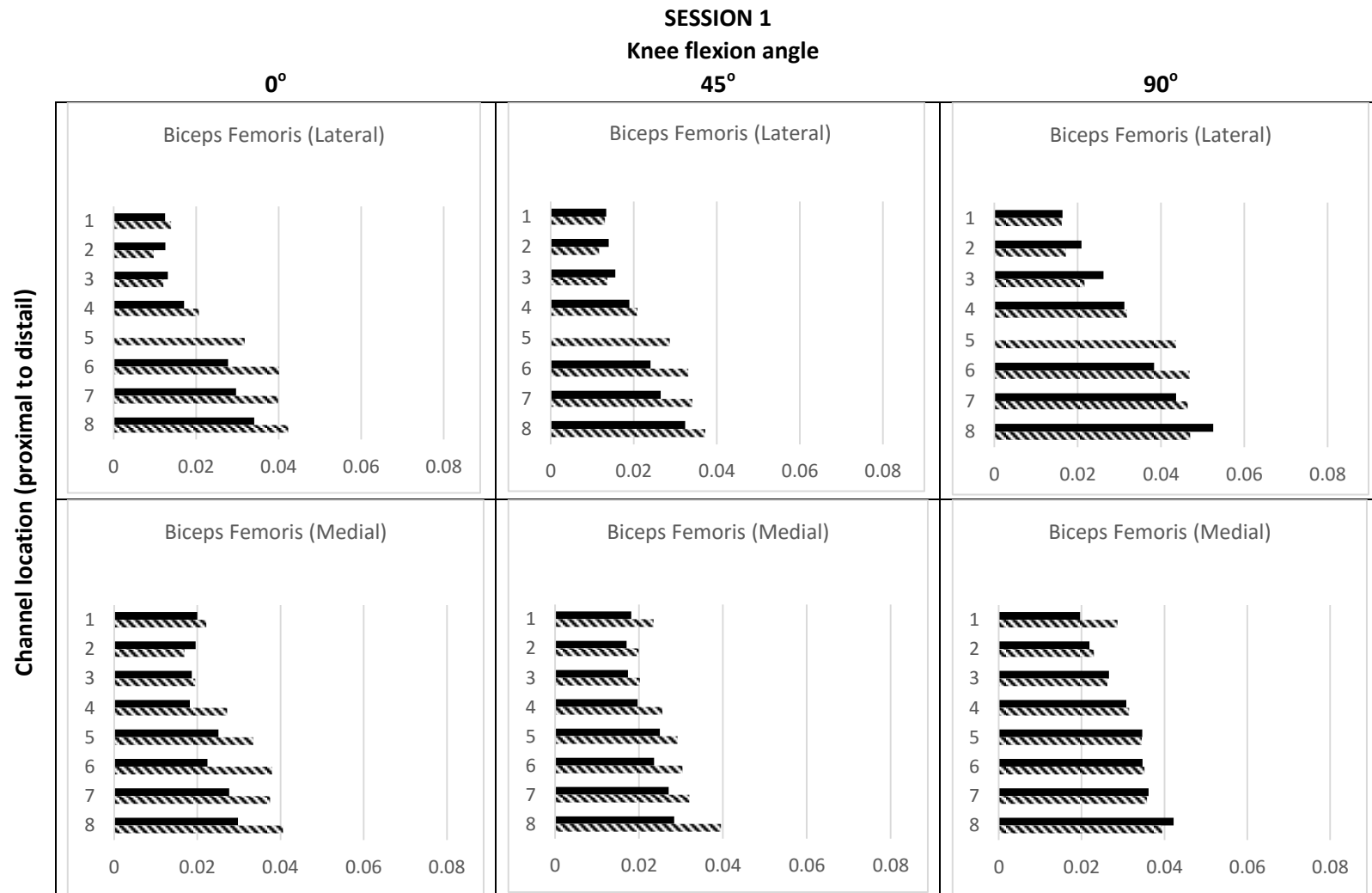


Figure 58: Channel contribution of the biceps femoris (medial and lateral) to maximal overall hamstring activity during each of three isometric contractions arranged according to knee flexion angle. Total activation =1. This data was collected during Session 1. Previously injured limb = black. Uninjured limb = hatched

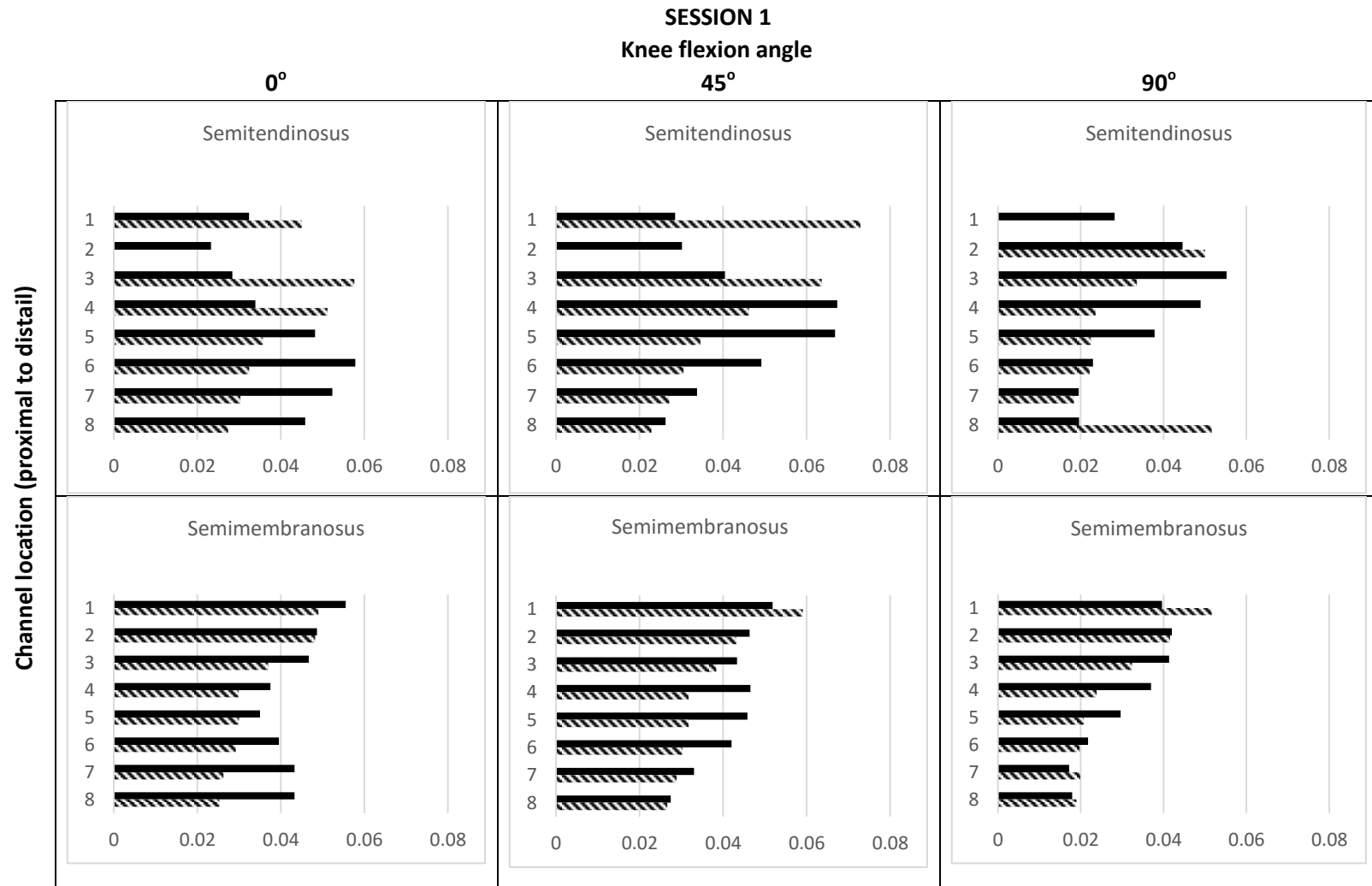


Figure 59: Channel contribution of the medial hamstrings to maximal overall hamstring activity during each of three isometric contractions arranged according to knee flexion angle. Total activation =1. The arrays of electrodes are nominally referred to as semitendinosus for the more lateral array and semimembranosus for more medial array. This data was collected during Session 21. Previously injured limb = black. Uninjured limb = hatched

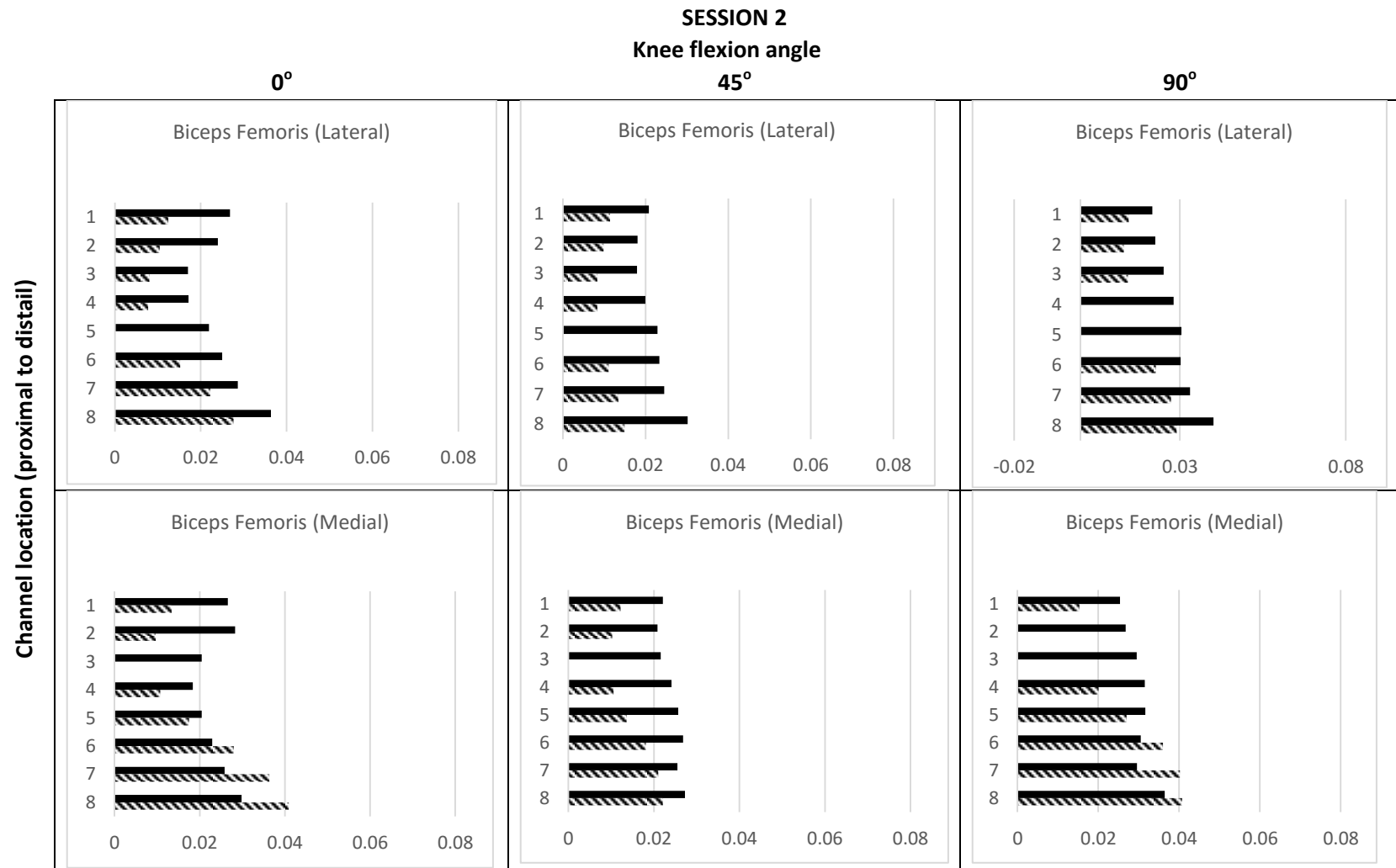


Figure 60: Channel contribution of the biceps femoris (medial and lateral) to maximal overall hamstring activity during each of three isometric contractions arranged in columns according to knee flexion angle. Total activation =1. This data was collected during Session 2. Previously injured limb = black. Uninjured limb = hatched

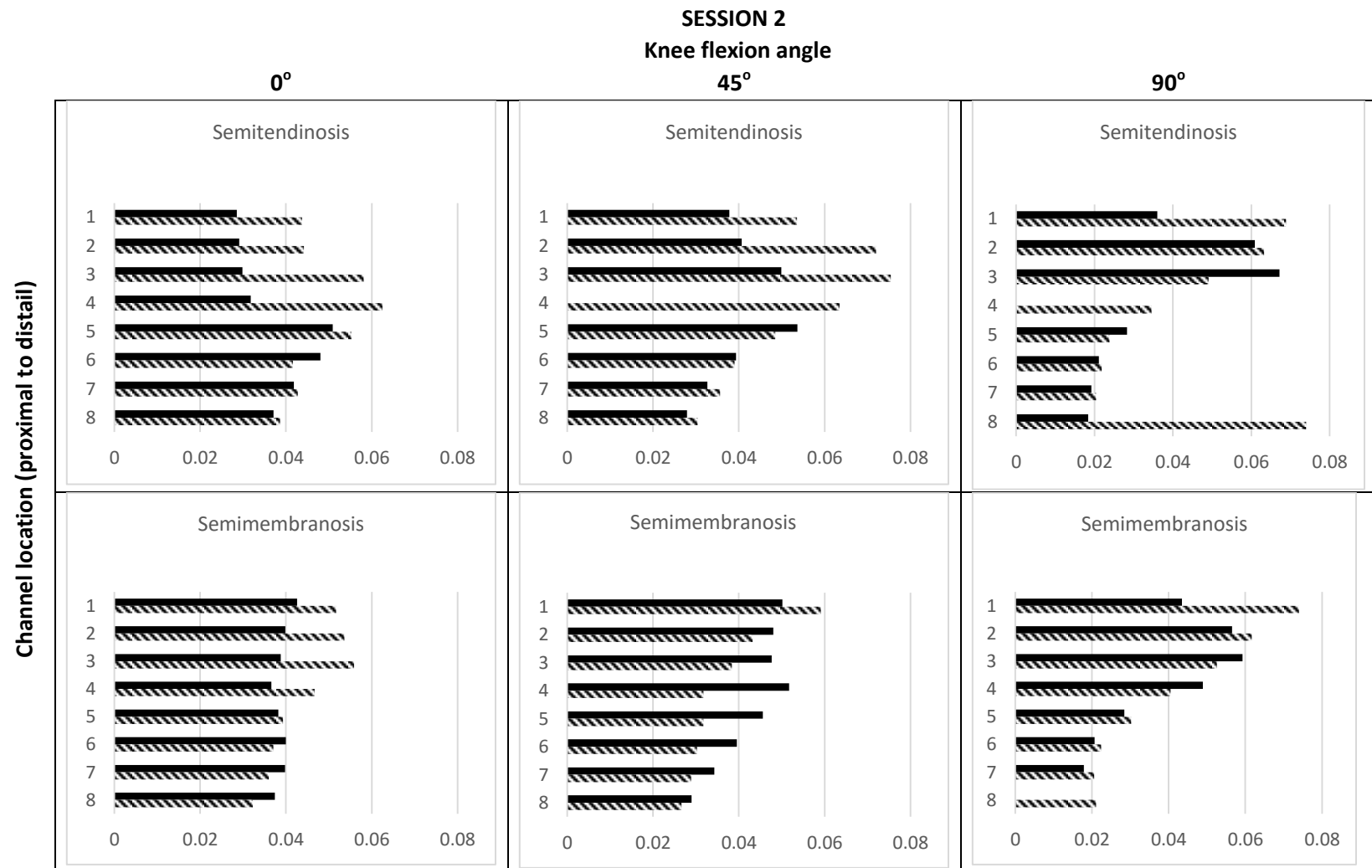


Figure 61: Channel contribution of the medial hamstrings to maximal overall hamstring activity during each of three isometric contractions arranged in columns according to knee flexion angle. Total activation =1. The arrays of electrodes are nominally referred to as semitendinosus for the more lateral array and semimembranosus for more medial array. This data was collected during Session 2. Previously injured limb = black. Uninjured limb = hatched

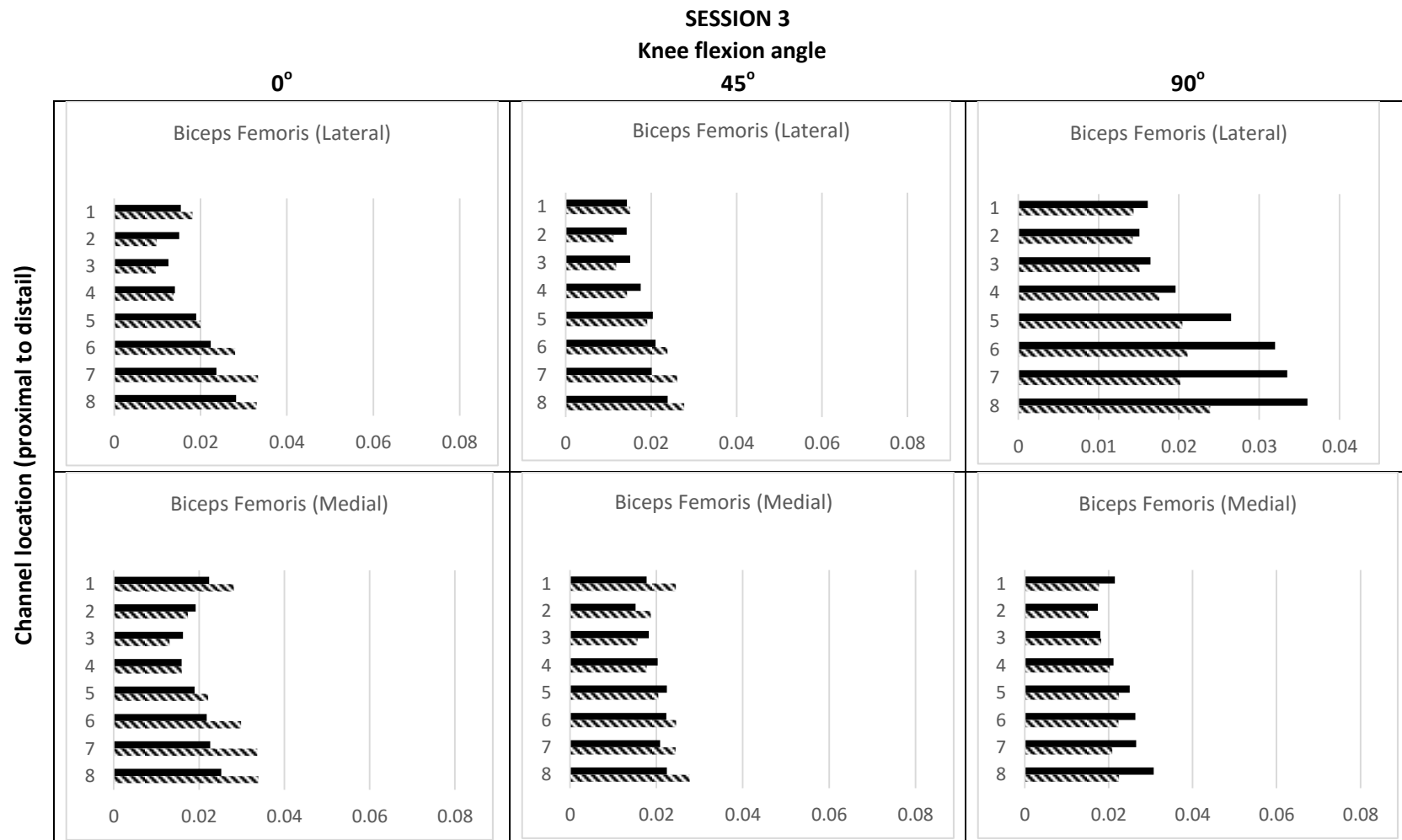


Figure 62: Channel contribution of the biceps femoris (medial and lateral) to maximal overall hamstring activity during each of three isometric contractions arranged in columns according to knee flexion angle. Total activation =1. This data was collected during Session 3. Previously injured limb = black. Uninjured limb = hatched

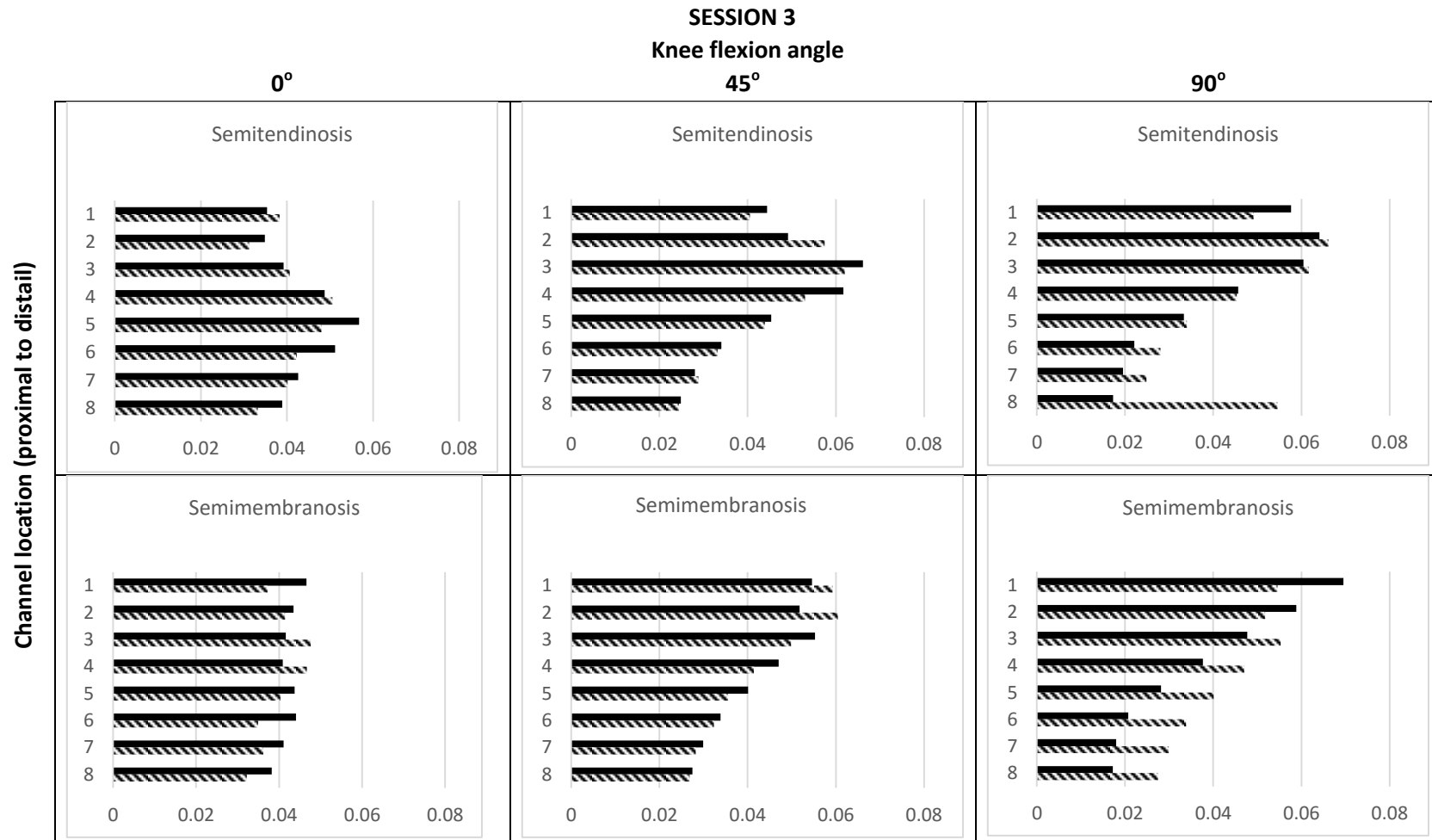


Figure 63: Channel contribution of the medial hamstrings to maximal overall hamstring activity during each of three isometric contractions arranged in columns according to knee flexion angle. Total activation =1. The arrays of electrodes are nominally referred to as semitendinosus for the more lateral array and semimembranosus for more medial array. This data was collected during Session 3. Previously injured limb = black. Uninjured limb = hatched

7.4 Discussion

In this chapter, measures of force generation and spatial hamstring muscle activation patterns during maximal effort contractions are reported in the early post-injury stages in two elite level athletes. These athletes present with quite differing profiles but with clear asymmetries apparent in both. Both athletes regained their ability to generate knee flexion forces to a similar (or greater) level when compared to their uninjured limb at the time of return to sport but the myoelectric asymmetries remained. Indeed, the sprinter (Case 1) was notably stronger on her previously injured limb during eccentric contractions when tested at return to sport. Spatial mapping of EMG signals may offer a novel, and clinically relevant insight into muscle function after acute injury. The association between altered spatial EMG activity and risk of future injury needs to be established in order to fully appreciate any predictive or screening potential of this approach.

The measurement of spatial EMG activity using low density, high surface array arrays over one muscle or muscle group, is mostly confined to measurements of the effects of fatigue and delayed onset muscle soreness (158, 161, 163). From this body of research, it is known that muscles display non-uniform response to fatiguing or damage inducing exercise. Differing areas of the same muscle appear to be uniquely affected by fatigue or delayed onset muscle soreness, a characteristic which may be linked to variations in fibre type concentration, muscle architecture and/or innervation patterns across the bulk of the muscle. No study to date has measured spatial activity following acute muscle injury.

The control of muscle recruitment originates in the supraspinal and spinal levels of the central nervous system and involves a complex interplay between a series of feedback and feedforward mechanisms (194, 195). This results in a controlled recruitment of motor unit pools across synergists with simultaneous inhibition of antagonists in order to produce a smooth and controlled movement proportional to the force required (135). These complex mechanisms are known to be adversely affected by peripheral inputs such as pain and proprioceptive afferent signals (153). These control mechanisms may also be altered by centrally mediated input such as motivation and apprehension to move (196). S-EMG offers a means by which these altered motor recruitment patterns can be detected. Indeed, altered patterns of activity have been measured in chronic low back pain (197, 198) as well as ligament injury about the knee (199).

Following acute muscle injury, nociceptive input secondary to acute inflammation may alter motor control in the early stages (153). However, both cases included here were free from pain during testing. The exception can be seen in the second test of the footballer, where the probable effects of delayed onset muscle soreness appear to have caused a slight irritation in the semimembranosus muscle as evidenced by oedema on MRI (see Figure 56). Indeed, the footballer was complaining of general discomfort in this region at that time but did not experience any discomfort specifically related to the testing. The possible manifestation of this suspected pain driven alteration in motor control may be seen in the reduced peak force values for the 45° knee flexion contraction when compared to the first test (Figure 57) and in the spatial activation graphs where reduced activity in the semimembranosus of the previously injured limb is noted (Figure 61). Interestingly, during this testing session, the previously injured biceps femoris appeared to be contributing more to overall activity when compared to the uninjured limb (Figure 60) perhaps indicating a compensation within the hamstring for impaired semitendinosus function.

The reduced ability for an injured muscle to generate force may also lead to alterations in recruitment patterns (153). Acute muscle injury repair is characterised by two simultaneous processes, the formation of a scar within the region of the muscle tear, alongside the ingrowth and regeneration of new muscle fibres from either side of the injury site. The healing tissues are vulnerable to injury as they have reduced ability to tolerate tensile load. Scar remodelling and the laying down of mature type 1 collagen, as well as continued regeneration of muscle fibres eventually leads to a restoration of force generating and tolerating capacity (29, 30, 56). It may be that neural activation patterns are altered to accommodate the reductions in force generating capacity about the site of injury. Increased recruitment of areas of uninjured muscle and/or adjacent and uninjured synergists may compensate for the injured areas. In the injured footballer, there appears to be reduced activation of the previously injured mid-distal biceps femoris during the first and third testing session (Figure 58 and Figure 62) as evidenced by a reduced contribution for these regions to overall maximal activity compared to the uninjured limb. In contrast, the medial hamstrings of the previously injured limb appear to show an increased magnitude of activation during the same test, perhaps suggesting a compensatory strategy. These patterns may signify a form of recruitment inhibition. Reduced biceps femoris

activity levels in previously injured athletes have been reported previously using both fMRI (68) and EMG (69, 70, 103).

The sprinters force and muscle function profile are markedly different to that of the footballer. She displays an ability to generate much greater levels of force in her previously injured limb during eccentric contractions (Table 14). She did display reduced strength on the previously injured limb during the 60° concentric contraction but this finding is at odds with the remainder of the isokinetic profile and was therefore deemed to be secondary to submaximal effort during this trial. Viewed in isolation, the sprinters isokinetic profile appears excellent. Croisier et al (32) provide prospective evidence that preseason strength imbalances detected with isokinetic testing are predictive for hamstring injury in the subsequent season. Specifically, strength imbalances of >15% , standard H:Q ratios of <0.47 and a mixed ratio of <0.87 were associated with future injury (RR 4.66. 95% CI 2.01-10.08). The sprinter meets none of these criteria and is, in fact stronger on her previously injured limb.

However, the sprinters previously injured biceps femoris shows notably elevated contributions to maximal activity when compared to the medial hamstring of the same limb and the biceps femoris of the opposite side (Figure 53 and Figure 54). This sprinter was supported as an elite athlete. Consequently, she was undergoing ongoing and intensive eccentric strength work both before and after she sustained her hamstring injury. Her isokinetic and EMG activation patterns seem to indicate an enhanced ability to generate eccentric force on the previously injured side and a propensity toward perhaps excessive levels of motor recruitment in the previously injured biceps femoris. Comparative excessive recruitment of the back extensors is reported in individuals with chronic low back pain (155, 200, 201). This feature is suggested to manifest secondary to maladaptive motor control strategies whereby the individual maintains excessively high level of motor activity through movement leading to nociceptive afferent activation and central sensitisation of the nervous system (155, 196). Although the sprinter did not report pain, the elevated contribution to maximal knee flexor effort by the previously injured biceps femoris may highlight a propensity to over-recruit this muscle. This in itself may lead to increase re-injury risk if the dependency on the biceps femoris during sprinting is excessive.

It is unknown if the sprinter's recruitment patterns are present secondary to intensive eccentric strengthening work during rehabilitation, or if they illustrate an alternative pattern of activation associated with prior hamstring injury. Post-injury neuroinhibition secondary to excessive scar formation, muscle atrophy and reduced optimal hamstring length has been proposed as underlying the elevated re-injury risk in athletes with previous hamstring injury (20, 61). The profile of the footballer seems to fit well with this theory, with areas of reduced contribution to maximal EMG activity seen in the region of the prior injury. The sprinter displays patterns of activity which do not suggest an inhibited activation about the site of the previous injury. Instead, the opposite appears to be the case and an over-recruitment about the area of the previous injury appears apparent perhaps suggesting an extension to the neuroinhibition theory may be required. However, analysis of much greater subject numbers would be required to quantify if the sprinter pattern is common or simply unique to this individual.

One further note is the contrast between the inner range (90°) and mid to outer range (0° , 45°) contractions in the footballer. Sprint related hamstring injury is thought to occur during late swing, a period when the hamstring is near the maximal functional length (72, 85). The footballer shows apparent under-activation of the mid-distal biceps in the mid-outer range but not in the inner range, indicating that the spatial activity alterations appear to be range specific.

There are a number of limitations in this analysis. Firstly, data is presented for two athletes and therefore generalisation across sporting populations is not possible nor advised based on these findings. Data related to spatial activation variance in the healthy hamstring muscles is limited but suggests consistent patterns of activation are apparent within and between subjects (184-186). Therefore, the interlimb asymmetries reported here are notable. In this study, descriptive observations are made and no statistical tests are performed. Nevertheless, this preliminary examination of post-injury hamstring function does highlight the potential use of low density, high surface area EMG as an assessment tool in the post-injury state. The sprinter lacked some familiarity with the isokinetic procedure. Although a series of trial runs preceded testing and a warm-up trial preceded each test, the subject may have lacked the skill to complete the assessment to the maximum of her ability as suggested by the data produced during the 60° /second concentric trial.

The spatial EMG analysis employed in the case studies is quite novel. We therefore lack an understanding of typical spatial recruitment in the hamstring. Previous research does suggest that spatial hamstring activity is repeatable in a healthy population (185, 186) but the study numbers are small. However, through employing such novel approaches in the assessment of post-injury muscle function, we may better understand the potential for detecting dysfunction using this approach. We may only surmise as to the clinical significance of the findings in this study but the asymmetries detected certainly highlight some interesting characteristics of post-injury muscle activity. The fact that these asymmetries continued to manifest at return to sport and following the restoration of force generating capacity suggest that low density, high surface area EMG mapping may offer enhanced insight into post-injury muscle function when compared to measures of force. Further research is need to determine population norms with this data and also determine the causal relation between altered activation patterns and future injury.

7.5 Conclusions

We present data relating to force generation capacity and spatial EMG activity in two athletes who had recently sustained a right biceps femoris injury during high speed running. Although force generation capacity appears restored following rehabilitation, both athletes presented with evidence of asymmetric recruitment patterns of their hamstrings at return to sport. The asymmetries detected were in direct contrast between each athlete with evidence of over recruitment about the site of the previous injury in one, and persistent under-recruitment about the injury site in the other. This data highlights the potential use of low density, high surface area EMG in the measurement of spatial muscle function following injury.

8 Discussion, future directions and conclusions

8.1 Main findings

The aim of this thesis was to explore the biomechanical characteristics of athletes who have returned to sport following sprint related hamstring injury. The thesis focussed on assessing various measures of force, movement and surface EMG signal in previously injured athletes. This exploration was completed firstly through summarising the already published data relating to post-injury biomechanics by means of a systematic review and meta-analysis, secondly by exploring lower limb kinematics and biceps femoris related EMG ratios during high speed running, thirdly by exploring spatial activation patterns and force generation during the Nordic hamstring exercise and finally through examining spatial activation patterns and force generation capacity during the early post-injury phases by means of two case reports. The review, presented in Chapter 3, indicates that previously injured athletes have particular deficits in their ability to generate slow eccentric knee flexor forces following return to play. Further, the running study, presented in 5, indicates that previously injured athletes run with movement patterns about the hip/pelvis which place the hamstring under increased length during the late swing phase. This is the period when the biceps femoris is at most risk of injury. Furthermore, EMG ratios values during running indicate patterns suggestive of impaired biceps femoris activation. Finally, as presented in Chapter 6, low density high surface area EMG, indicated that the proximal previously injured hamstring appears less active during Nordic loading, while the medial hamstring median frequency values appear reduced suggesting general inhibition of proximal and medial motor unit recruitment on the previously injured side. However, the case reports, presented in Chapter 7, suggest that alteration in spatial activation patterns may differ between individuals. This may be in response to rehabilitation or indicative of sub-categories of post-injury dysfunction. The overall content of this thesis is summarised in Figure 64.



Figure 64: Infographic summarising the content of this thesis.

8.2 Measuring recovery and determining recurrence risk following hamstring injury.

For rehabilitation to be successful following hamstring injury, two key outcomes must be achieved. Firstly, the athlete must be, at least returned to their pre-injury levels of function. Secondly, the risk of re-injury must be minimised in so far as possible. In attempting to reduce the risk of injury recurrence one must first be able to detect and address any modifiable risk factor in the previously injured athlete. This is especially true in the case of hamstring injury, where previous injury is the leading risk factor for future injury accounting for up to an 11.6 fold increase in risk (OR, 95% CI 3.5-39.0) (17, 18, 20, 202). However, it has yet to be determined which characteristics in the previously injured athlete are responsible for the increased recurrence risk (20).

Current approaches to the assessment of an athlete following hamstring injury are heavily reliant on radiological imaging. An example of this can be seen in the case of the footballer in Chapter 7, where repeat MRI scans were undertaken to monitor recovery. Several competing MRI classification systems exist (27, 80, 81, 203) all of which are broadly based on the O'Donoghue classification reflecting increasing levels of structural damage to muscle fibres (204). As imaging technology has advanced, higher resolution images have enabled radiologists to report which specific components of the muscle architecture are damaged, at which location and to what degree. Indeed the classification systems proposed by Chan et al (80) and Pollock et al (81) suggest muscle injuries should be sub-classified into as many as 12 subtypes depending on location and severity. However, the location and magnitude of a muscle fibre damage only represents the most peripheral and gross consequence of the injury. Indeed, the value of imaging in determining restoration of function and measuring recurrence risk following hamstring injury has only been established in a most basic manner. A recent systematic review by Reurink et al (205) found that research in this area is predominantly of poor methodological quality. They report moderate evidence suggests that an absence of changes on MRI following hamstring injury is associated with shorter times to RTP, while injuries involving the proximal free tendon are associated with a longer time to RTP. In addition, they report limited evidence indicating longer RTP times for athletes with total ruptures, disruption to the central tendon and injuries not affects the musculotendinous junction. Furthermore, it is recognised that up to 42% of athletes continue to display residual abnormal signal intensity on MRI at RTP. There is minimal

evidence of an association between MRI features and risk of recurrence (206, 207) with indications that larger volume strains tend to recur (11, 96, 208, 209). Consequently, MRI imaging appears to have limited clinical applications beyond providing a description of the location and severity of the lesion. Current evidence suggests that this information is of limited clinical relevance with specific regard to monitoring and predicting functional recovery, and in determining re-injury risk (43, 98, 210).

De Vos *et al* (43) report prospective evidence that clinical measures of discomfort on palpation (AOR 3.95; 95% CI 1.38 to 11.37), isometric knee flexion force asymmetries (AOR 1.04; 95% CI 1.01 to 1.07) and an active knee extension deficit (AOR 1.13; 95% CI 1.03 to 1.25) are associated with increased injury recurrence risk. This information is based on a cohort of 64 amateur athletes from varying sports who were assessed after RTP. Critical analysis of these findings indicate that the adjusted odds ratio for the force and flexibility outcomes are moderate at 1.04 and 1.13 respectively. Indeed, the magnitude of asymmetry detected in the active knee extension test was clinically small at -2° (SD 5°) while the difference in peak force was -2N (SD 15N). Certainly the presence of pain on palpation was associated with close to a 4-fold increase in recurrence risk, but such a characteristic seems to imply incomplete rehabilitation. Indeed, subjects were excluded from the studies outlined in Chapter 5 and 6 if they presented with this characteristic.

The majority of research exploring clinical and biomechanical features present in previously injured athletes is retrospective in nature. This includes the research reported in this thesis. Retrospective design does not allow for the determination of causality i.e. one cannot determine if a measured deficit was present in advance of the injury. However, in the absence of prospective data on hamstring injury recurrence risk factors, a detailed analysis of the previously injured athlete may highlight characteristics worth consideration for future prospective research.

8.3 Force related outcomes following hamstring injury.

The systematic review presented in Chapter 3 highlights that previously injured athletes appear to present with persistent deficits in strength particularly during slow eccentric type contractions. Maniar *et al* (211) recently published a systematic review and meta-analysis of strength measures and flexibility in athletes who had previously sustained a hamstring injury. In contrast to our approach, they include athletes in the pre and post return to

sport phases. Interestingly, they found that although deficits in isometric strength were apparent in the early stage of rehabilitation, these had resolved at the time of return to sport. In agreement with our findings, Maniar et al (211) found variable evidence of isokinetic and Nordic loading strength deficits persisting beyond return to play. It seems possible that strength measurement during eccentric loading may be sensitive in detecting post-injury hamstring dysfunction. Interpretation of the research base is made more complex by the wide range of approaches to isokinetic testing. Nevertheless, maximal effort eccentric loading may expose persistent deficits in force generating capacity in some previously injured hamstrings in a way that isometric or concentric effort contractions do not. However, this finding is not universal. We failed to detect any significant difference in force generation capacity between previously injured and uninjured/control limbs in our Nordic loading experiment in Chapter 6. Similarly, in the systematic review in this thesis, presented in Chapter 3, when analysis is limited to elite level athletes, there are no deficits in Nordic force outcomes noted.

Eccentric loading is associated with greater levels of motor unit recruitment, increased torque output and more cortical involvement than concentric or isometric contractions (74, 75, 122). The implication based on this thesis, is that maximal eccentric load may, in some cases, may be more sensitive in detecting post-injury hamstring dysfunction. It may be that near total activation of available motor units is required to expose deficits. This highlights that perhaps post-injury dysfunction is not isolated to the muscle fibres. It may be that the aetiology of post-injury force generation deficits may originate systemically, or lie at the spinal or supra-spinal level and involve the neural control of motor unit recruitment. Indeed, there is some functional MRI evidence to suggest that post injury inhibition is not simply confined to the previously injured muscle but can involve the adjacent muscles of the hamstring group (68). In Chapter 6 of this thesis, alterations in spatial activity were detected during the Nordic when comparison was made to control group limbs, but not to the uninjured limbs of the previously injured subject. Therefore, one may tentatively infer that the uninjured side was also altered versus the control limb indicate perhaps some bilateral manifestations of dysfunction. This perhaps indicates centrally or systemically mediated alterations in function rather than changes simply existing in the periphery. This point is considered below.

8.4 Movement outcomes following hamstring injury

The assessment of hamstring function during high speed running may offer more detailed and functional insight into post-injury biomechanical dysfunction. The biceps femoris injures during late swing in sprinting. This is a period of maximal eccentric activity, while the muscle is in a lengthened and biomechanically disadvantageous position (45, 72, 85). The biomechanical disadvantage may be magnified by altered the position of the pelvis during this phase. Increased anterior pelvic tilt lengthens the biceps femoris via its proximal attachment to the ischium (24). The position of the pelvis can be influenced by increased or decreased activity in a range of muscle which attached around the pelvis. Indeed Chumanov et al (46) have highlighted the potential influence of muscles about the pelvis on biceps femoris length during terminal swing. Furthermore, rehabilitation programmes which incorporate postural control work are associated with reduced re-injury rates (62, 63). The systematic review in this thesis identified three studies which measured 3D movement patterns during running (103, 116, 119). One of these studies (103) was conducted by our group and is reported in Chapter 5. Meta-analysis was not possible due to contrasting statistical methods employed. Silder et al (119) inputted their data into a musculoskeletal model designed to measure hamstring length, finding no significant differences between injured and controls during late swing. Both our study (103) and Lee et al (116) report significant differences in late swing movement in previously injured limbs. However, the findings are contrasting with our study indicating patterns suggestive of increased anterior tilt/hip flexion on the injured limb, and Lee et al (116) finding the opposite. However, Lee et al (116) and Silder et al (119) made comparisons using quite a diverse study population limiting the generalisability of their findings. We, in contrast, constrained our analysis to a single sport and level, paying particular attention to matching between the study groups. Nevertheless, it is acknowledged that our numbers are small (n=17) and therefore caution should be used when attempting to generalise our findings.

8.5 EMG outcomes following hamstring injury

The systematic review outlined in Chapter 3 indicated that there is almost universal agreement that the previously injured biceps femoris displays reduced amplitude activity levels when comparison is made to uninjured limbs or functionally relevant muscle pairs. This reduction in activity is apparent during eccentric isokinetic testing (69, 70, 108). Our

study, included in both the systematic review and written in chapter form (Chapter 5) indicates relatively reduced biceps femoris activation on the previously injured limbs during late swing compared to control subjects.

Silder et al (119) found no difference in EMG amplitude of the biceps femoris based on an inter-limb analysis of previously injured participants. The findings from Silder et al (119) appear to contradict the findings from our research (103). However, Silder et al (119) made comparisons to the uninjured limb of the previously injured subject. This approach may have confounded their approach. This point is more clearly made when one considers the findings relating to the low density, high surface area spatial EMG measures in Chapter 6. This study revealed no significant difference between the limbs of previously injured subject but significant alterations in spatial activity when comparison is made to controls. Perhaps the manifestation of EMG alterations associated with prior hamstring injured are not side or site specific. This is further supported by the finding that the contribution ratios to maximum activation was reduced in the proximal aspect of the previously injured muscle, while the median frequency was altered in the medial hamstrings. The injury site is likely to be the mid-distal biceps (189) but the alteration noted are remote to this area. Bourne et al (68) report similar findings with reduced global activation of previously injured hamstrings during the Nordic exercise when relaxation times are measured with functional MRI. This again highlights that post-injury hamstring dysfunction is complex and appears to involve the organisation motor unit activation throughout the hamstring group and perhaps in the contralateral limb.

8.6 The aetiology of hamstring related dysfunction following injury.

There is growing recognition that previously injured athletes display characteristics which cannot simply be attributed to the structural consequences of the muscle injury. The high recurrence rates and persistence of functional deficits following hamstring injury indicates a chronic after effect of the injury. The neuroinhibition theory proposed by Fyfe et al (61) certainly illustrates a potential mechanism whereby suboptimal healing results in chronic maladaptation a resultant increased re-injury risk. These authors propose that excessive scar tissue and shortened muscle fascicle lengths cause biomechanical disadvantages in the post-injured state. Certainly, persistently increased scar volume has been reported in previously injured athletes (28) and shorter biceps femoris fascicle lengths has been

reported as a strong risk factor for first time hamstring injuries (RR=4.1; 95% CI 1.9 to 8.7) (212). However, neither of these features have been assessed in relation to injury recurrence risk.

The data presented in this thesis, as well as the functional MRI data presented by Bourne et al (68) suggests that previously injured athletes display evidence of dysfunction beyond the site of the muscle lesion, even in the contralateral limb and muscles elsewhere about the pelvic girdle. This suggests that aspects of post-injury hamstring dysfunction may be driven centrally or systemically. Pelletier et al (213) propose that maladaptive neuroplastic responses to injury may underlie the chronic dysfunction seen in some musculoskeletal disorders. With particular reference to painful conditions e.g. chronic low back pain, patellofemoral pain syndrome, osteoarthritis and carpal tunnel syndrome, these authors outline several experiments which measured functional changes at the spinal, brain stem, sensory and motor cortices and limbic system levels. These changes may result in behavioural alterations, perceptual changes and altered sensory-motor control. It is certainly worth considering if similar features manifest following hamstring muscle injury.

Athletes over the age of 24 are at increased risk of hamstring injury (15, 20, 95, 123, 202). Only one study has examined the reasons for this feature suggesting hip flexor tightness may be a contributing factor. Indeed, the data presented in Chapter 5 of this thesis indicates that athletes who have sustained an injury running with increase anterior tilt during late swing, a feature which may be driven by contralateral hip flexor tightness. Furthermore, perhaps the non-lesion specific nature of post-injury hamstring dysfunction indicates a metabolic driver of dysfunction. Muscle development, repair and regeneration is driven by cytokine and hormonal influences (214-216). Age related changes in hormone levels have not been examined in relation to hamstring injury or hamstring function in athletes. Future research in this area may yield clinically relevant findings.

8.7 Limitations

The studies presented in this thesis are limited by their retrospective nature. Although efforts were made to ensure close matching of experimental groups, we cannot determine if the alterations detected in previously injured subjects existed prior to, or as a result of their injury. For this reason, no causal relationships can be identified with confidence.

Despite concerted effort to recruit participants, our final subject numbers are low. Furthermore, case report data is presented in Chapter 7 which represents level 5 evidence

and any generalisation of this data to wider populations is not advised. Nevertheless, our subject numbers are in agreement with other similar published biomechanical research and provide clinically relevant insight into biomechanical characteristics following hamstring injury. Although effect size calculations were used to ensure minimisation of the risk of type 1 and 2 error, further research is required to confirm these findings in larger population samples. Until then, caution should be employed if attempting to generalise the findings across other sports and individuals.

Finally, the more novel aspects of the data collection and analysis approaches in this thesis provide enhanced insight into post-injury biomechanics. However, these approaches are uncommon in the wider research context. Therefore, knowledge about normal variation in healthy participants was lacking. This limitation was countered by making comparisons with matched control subjects, however caution is advised as the control population sample size was small and may not have been totally representative of an uninjured population. Increasing the sample size of the control group dataset would establish the expected variance across an uninjured population and allow for more trustworthy comparison.

8.8 Clinical impact

There is growing recognition that hamstring injury is complex and multifactorial in nature. Altered neuromuscular activation patterns appear to manifest for a prolonged period after injury and may underlie the high re-injury rates seen with this injury. Current rehabilitation strategies appear to be inadequate at preventing reinjury and rates of hamstring injury have increased over recent years (13, 14). Furthermore, the use of EMG ratio analysis, alongside 3D motion capture appears to illustrate characteristic of function which may place the previously injured muscle at increased length and increase re-injury risk. The methods described in this thesis may therefore offer valuable insight for the treating clinician. Should a causal relationship be established between the post-injury characteristics highlighted in this work and future injury risk, these approaches will become valuable screening and rehabilitation tracking tools.

This thesis highlights novel approaches to post-injury biomechanical assessment. In particular, the measurement of muscle activity during function may offer enhanced insight compared to static measures of structure such as MRI. In particular, the use of low density, high surface area EMG appears to illustrate alterations in muscle activation which remote

to the muscle injury site. The non-lesion specific nature of hamstring related dysfunction challenges the assessing clinician to consider issues beyond the structural manifestations of the injury. Adequate rehabilitation and successful reduction of re-injury rate may involve retraining of movement patterns and the perceptual maladaptations which either manifested before or as a result of the injury. The influence of hormonal and physiological sequelae of injury is unknown but may highlight further areas requiring intervention.

8.9 Future directions

Follow up testing with larger subject numbers is required to firmly establish the prevalence of the characteristics described in this thesis in athletes with previous hamstring injury. If low density, high surface area EMG prove sensitive in detecting apparent compensatory motor activity following hamstring injury the development of more user friendly detection hardware is warranted. The methods employed in Chapter 6 enabled a highly detailed examination of spatial activity, but were complex and laborious in their application. Linear array electrodes have been developed by Merletti et al (taken from Zwarts et al (134)). Modification of such sensor arrays for use in the sporting context would be welcome.

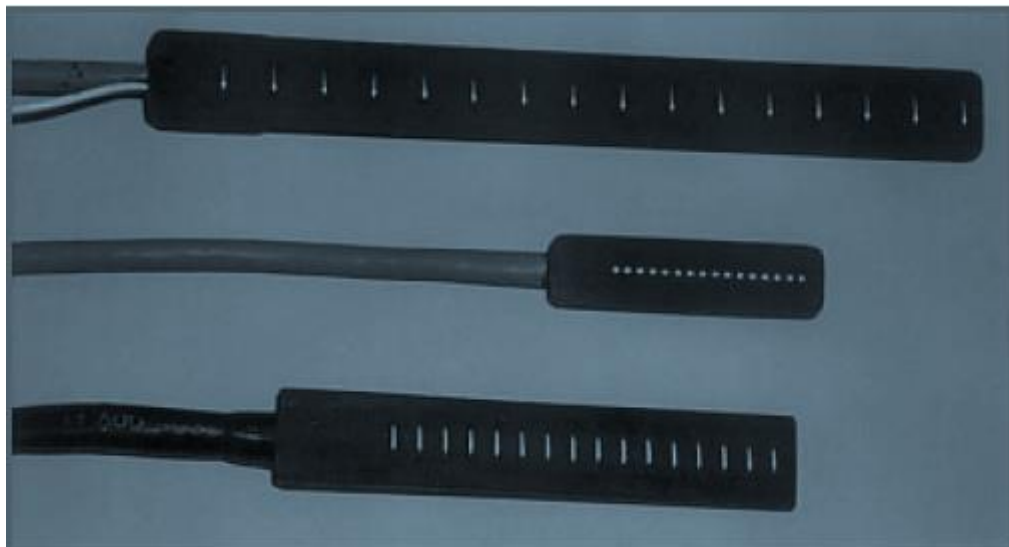


Figure 65: Linear EMG arrays by Merletti et al, taken from Zwarts et al (134).

The clinical relevance of the projects outlined in this thesis is to highlight characteristic of post-injury dysfunction. Nevertheless, the clinical impact of this work is dependent on prospectively linking the presence of such characteristics with the risk of future re-injury Therefore further prospective observational research will be required to prospectively

measure the causal relationship between identified deficits on re-injury risk. If a causal relationship is identified, interventions specifically designed to address deficits in function will need to be developed and their effectiveness both in terms of ameliorating the deficits and reducing the injury risk established.

Finally, the widespread neuromuscular changes noted to be present in athletes following hamstring muscle injury suggest inhibition and compensatory muscle activation patterns. An investigation of the central nervous system drivers underlying this dysfunction is warranted. In stroke research transcranial magnetic stimulation is often employed to measure motor responses to motor cortex stimulation. Perhaps such methods may provide further insight into sports related neuromuscular inhibition and guide the development of enhance assessment and rehabilitation strategies.

8.10 Conclusions

The projects outlined in this thesis provide clear evidence of persistent deficits in post-injury hamstring related muscle function following return to sport. The novel methodology offer new insight into post-injury muscle function. This research strongly indicates that the assessment of post-injury hamstring muscle function, must move beyond structural assessment. Consideration must be given to muscle activity during functional tasks. Although this is complex, this research highlights that alterations in muscle function are not localised to the injury site. Issues such as generalised hamstring muscle inhibition, compensatory recruitment of other pelvic girdle muscles and potentially injurious movement pattern alterations appear to persist after return to sport.

Given the persistently high prevalence and recurrence rate seen with sprint related hamstring injuries in sport, there is need to develop enhanced screening tools to understand and track post-injury muscle function. Movement data, EMG ratio data and low density, high surface area EMG collecting during hamstring dominant functional tasks, may provide key information related to post-injury dysfunction. These approaches may offer useful screening potential and provide a basis to develop and track enhance rehabilitation approaches, the ultimate goal of which will be to reduce recurrence rates.

9 Appendix

9.1 Anatomy of the hamstring

The hamstring is comprised of three bi-articular muscles (the biceps femoris long head, the semimembranosus and the semitendinosus) and the uni-articular biceps femoris short head. The origin of the bi-articular hamstring muscles is about the ischial tuberosity of the pelvis. The proximal tendon of the biceps femoris (long head) and semimebranosus are intimately related to one another with some fibres of the biceps femoris being continuous with the sacrotuberous ligament of the pelvis. The semimembranosus tendon originates more lateral and superior on the tuberosity, its tendon passing deep to the biceps femoris (long head) in a lateral to medial direction. The position of these attachments means that proximally, the hamstring muscles can function concentrically to both extend the hip and tilt the pelvis posteriorly. Conversely, eccentrically the hamstrings act to control a flexing hip and/or anterior tilting pelvis. The biceps femoris (short head) originates along the linea aspera, lateral supracondylar line and the intramuscular septum separating vastus lateralis from the biceps femoris (long head) (60, 217).

The anatomical arrangement of the distal attachments of the hamstrings suggest that their action is more than the simple concentric action of knee flexion and the eccentric control of knee extension. The medial and lateral insertions wrap around the knee joints and enable control of tibial/knee joint rotation (218-220). The distal biceps femoris (long and short head) tendon communicates via several slips with the lateral condyle of the femur, lateral collateral ligament (making it taut on contraction), fibular head, tibiofibular ligaments, popliteus tendon (and lateral meniscus), the arcuate ligament and crural fascia. The distal tendon of the short head is indistinguishable from that of the long head (219). Dissection of the distal attachment of the biceps femoris leads to antero-posterior and rotational instability of the knee joint (218). Medially, the semimebranosus has a broad, long, aponeurotic distal tendon which inserts along the medial tibial condyle, communicating with the popliteal region. The semitendinosus has a long distal free tendon (~11cm) which wraps around medial aspect of the knee, and, along with sartorius and gracilis, forms the pes anserinus insertion on the antero- medial aspect of the tibia. Onward connections to the pes anserinus, crural fascia and medial collateral ligament

enable this muscle (along with semimebranosus, sartorius and gracilis) to act as a strong medial stabiliser of the knee both in the sagittal and transverse plane (217, 220).

The intramuscular portion of the proximal tendon of the biceps femoris (long head) extends along nearly half the length (46.8%) of the bulky muscle belly. It is along this anatomical interface that the majority of sprint related hamstring injuries occurs. Distally the intramuscular portion of the distal tendon again begins high in the muscle, with the distal MTJ running along the lower 41% of the belly (217).

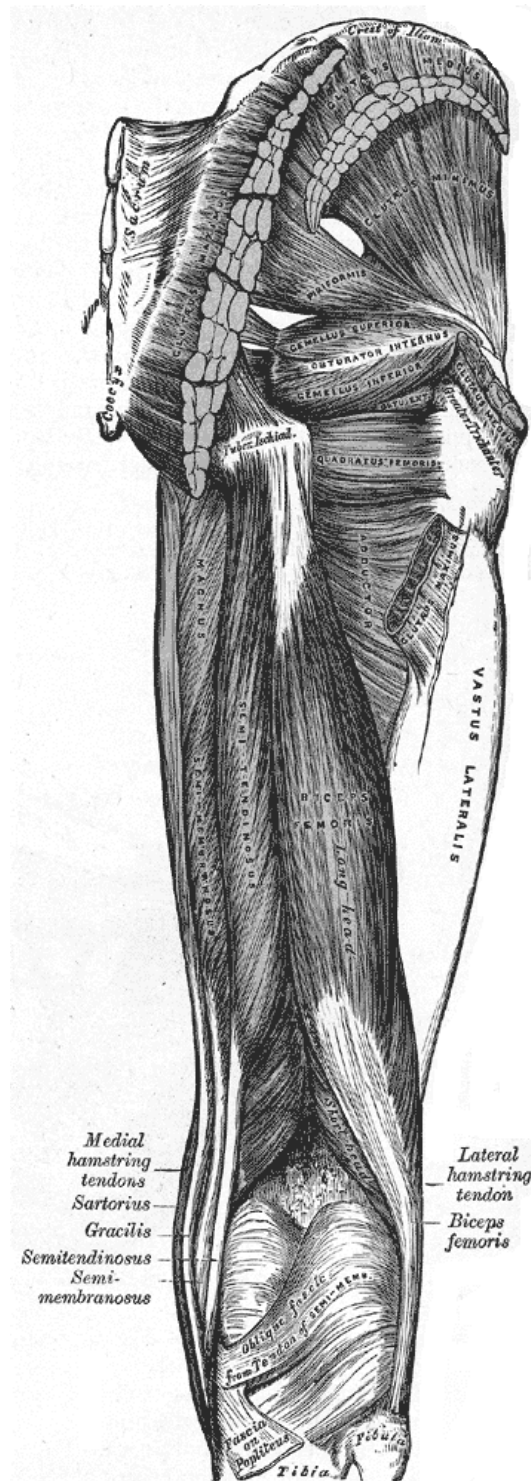


Figure 66: Anatomy of the posterior thigh.(221)

9.2 Matlab analysis programmes

9.2.1 Running Data (Chapter 5)

9.2.1.1 Kinematic Analysis

9.2.1.1.1 HamsMarkStrides.m

This programme is used to segment the raw kinematic data according to heel strike and toe off for both the ipsilateral and contralateral limbs. The process involves recalling the listed records to mark the dataset. The resulting marks are HSI & TOI for the ipsilateral side and HSC & TOC for contralateral side. These variables are appended to the coda file for each record. The code and outputs are outlined in the following sections

9.2.1.1.1.1 Identifying the heel and toe markers

The code in this section is used to select the relevant markers used to identify heel strike and toe off. These correspond to the marker on the calcaneus (heel) and 5th metatarsal (toe) on each foot i.e. markers 11 and 12 on the left and on the markers 21 and 22 on the right. In order to ensure that the left or right side is identified as the ipsilateral (i.e. previously injured) or contralateral (i.e. uninjured) the section uses the GetFileName function to designate the sides according to injury. The 'L' designation indicates a left sided injury and the 'R' a right sided. The resulting outputs are:

- IHeel (ipsilateral heel marker)
- CHeel (contralateral heel marker)
- IToe (ipsilateral toe marker)
- CToe (contralateral toe marker)

```
% clear all sections and select the running speed for analysis (i.e. 10, 15, 20 or ''  
for the reliability data)  
clear  
Speed='20';
```

```
% This loop is used to identify the heel and toe markers for the ipsilateral  
and contralateral limbs for all 18 subjects.
```

```
for S=1:18;
```

```
    [CodaFile, Subj, LR]= GetFileName(S, Speed);
```

```
% This is the switch which identifies the correct markers according to the  
contralateral (CL) limb as determined by the GetFileName function
```

```

if LR=='L';
    CL='R';
    IHeel=11;
    CHeel=21;
    IToe=12;
    CToe=22;
else
    CL='L';
    IHeel=21 ;
    CHeel=11;
    IToe=22;
    CToe=12;
end

```

9.2.1.1.1.2 Save Heel Strike and Toe Off data to the CodaFile

In this section Heel Strike and Toe Off data is stored as IHeelDat and IToeDat for the ipsilateral (i.e. previously injured) limb and CHeelDat and CToeDat for the contralateral (i.e. uninjured) limb.

```

% Clear the argument inputs
IHS=[]; CHS=[]; ITO=[]; CTO=[];

```

```

% Load the associated data from the CodaFile (kinematic data set)
load(CodaFile, 'MarkerData', 'MarkerNames', 'MarkerOcc', 'MarkerTime',...
'SegRotData', 'SegRotNames', 'IHS', 'CHS', 'ITO', 'CTO');

```

```

% Create Ipsilateral data via the MarkStrides function and display
IHeelDat=squeeze(MarkerData(:,:,IHeel));
IToeDat=squeeze(MarkerData(:,:,IToe));
disp([int2str(S) ' : ' CodaFile ' Ipsilateral']);
Title=[Subj ' Ipsi'];
[IHS, ITO]=MarkStrides(IHeelDat, IToeDat, IHS, ITO, Title);

```

```

% Create Contralateral data via the MarkStrides function and display
CHeelDat=squeeze(MarkerData(:,:,CHeel));
CToeDat=squeeze(MarkerData(:,:,CToe));
disp([ CodaFile ' Contralateral']);
Title=[Subj ' Contra'];
[CHS CTO]=MarkStrides(CHeelDat, CToeDat, CHS, CTO, Title);

```

```

% Save data as time points to the CodaFile for each subject in the subject
CodaFile
save(CodaFile, 'IHS', 'ITO', 'CHS', 'CTO', '-append')

```

Example Output for subject 05H04:

File Name/Location:

C:\Subject Raw Data\05H04\CODA\20kph.mat

Side:

Ipsilateral

Heel Strike Time Points:

0.7615 1.3807 2.0046 2.6193 3.2431 3.8532 4.4633 5.0872 5.6972 6.3073
6.9220 7.5596 8.1743 8.7936 9.4174

Toe Off time points

0.9372 1.5596 2.1697 2.7982 3.4037 4.0138 4.6284 5.2661 5.8853 6.4954
7.1009 7.7363 8.3349 8.9633

File Name/Location:

C:\Subject Raw Data\05H04\CODA\20kph.mat

Side:

Contralateral

Heel Strike Time Points:

0.4633 1.0734 1.6972 2.3073 2.9266 3.5550 4.1697 4.7890 5.3945 6.0229
6.6239 7.2385 7.8716 8.4862 9.1055

Toe Off time points

0.6450 1.2683 1.8779 2.4966 3.1055 3.7339 4.3532 4.9587 5.5780 6.2064
6.8094 7.4220 8.0596 8.6651

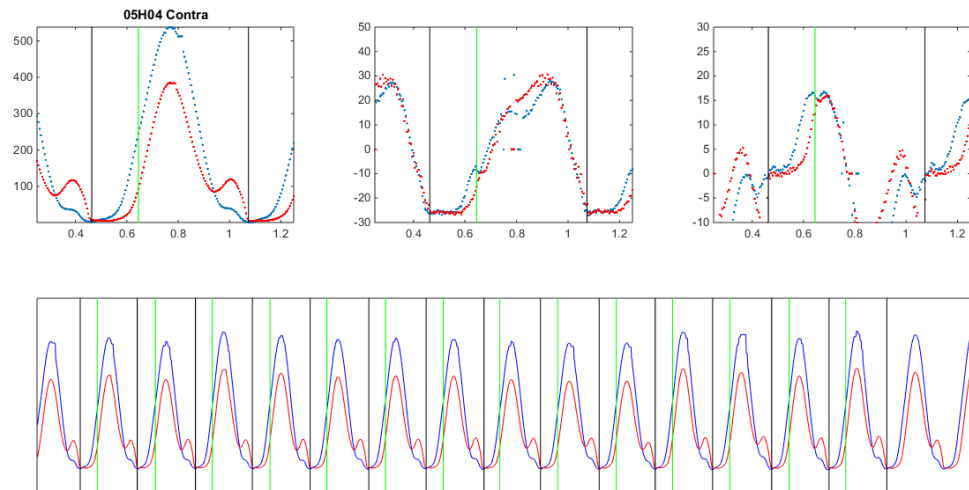


Figure 67 Graphic produced by MarkStrides function showing the graphics user interface for the contralateral data (Subject 05H04). The data is arranged as vertical position of heel and toe markers for one stride (top left window), horizontal velocity of the heel and toe markers for one stride (top middle window), vertical velocity of the heel and toe markers for one stride (top right window) and vertical position of heel and toe markers for all strides recorded (bottom window). The heel marker is in blue and the toe marker is in red while toe off is marker with the green vertical line and heel strike with the black vertical line. The vertical lines on place on the graphic with a right a left click of the mouse and can be positioned using a crosshair on any window.

end

9.2.1.1.2 MarkStrides Function

This function creates a Graphic User Interface or GUI which displays heel and toe marker kinematic data upon which heel strike and toe off times can be determined across the entire data set.

The outputs of this function are:

- Heel strike time points for both feet (HS)
- Toe off time points for both feet (TO)

The inputs for this function are:

- Kinematic data related to the heel markers (HeelDat)
- Kinematic data related to the toe markers (ToeDat)
- Any pre-saved heel strike data (HS)
- Any pre-saved toe off data (TO)
- The title of the graph from the HamsMarkStride script (Title)

This is summarised as follows:

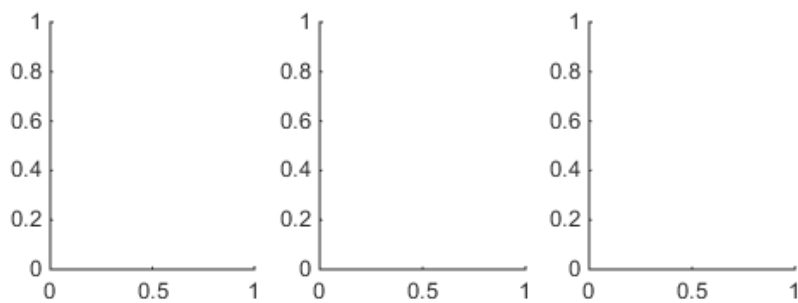
```
function [HS TO]=MarkStrides(HeelDat, ToeDat, HS, TO, Title)
```

9.2.1.1.2.1 Section 1

This first section outlines the layout of the figure for plotting heel strike and toe off data.

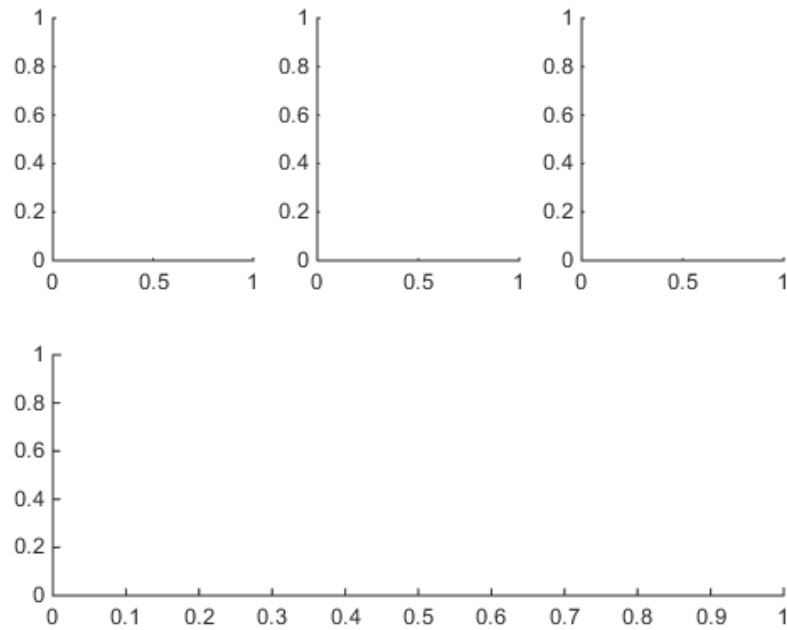
Initially 3 small subplots (hh(j) where j=1:3) are created across the top of the figure

```
for j=1:3; hh(j)=subplot(2,3,j); cla(hh(j), 'reset'); end;
```



Next a large subplot (hh(4)) is created across the bottom of the figure

```
hh(4)=subplot(2,3,4:6); cla(hh(4), 'reset');
```



```
drawnow
```

The 'x axis' of each plot equates to time and is laid out in seconds (i.e. the length of `HeelDat` which is collected at 200hz divided by 200).

```
Time=(1:length(HeelDat))/200;
```

9.2.1.1.2.2 Section 2

Within this second section `MData` is created. This is a concatenation of the kinematic data relating to the heel markers (`HeelDat`) and toe markers (`ToeDat`) across the third dimension, thus creating a series of cell arrays for each marker across time.

```
MData=cat(3,HeelDat, ToeDat);
```

9.2.1.1.2.3 Section 3

This third section plots heel and toe data within each of the defined subplots according to various conditions.

First, a series of elements are defined for later use.

```
Heel=1; Toe=2;
but=1; show=50:250;
```

All figures are reset and the `Title` is plotted above subplot `hh(1)` in the top left of the figure

```
while ~isempty(but for j=1:4; cla(hh(j),'reset'); end
    title(hh(1), Title)
```

One second of data around each HS and TO is defined as `useHS` and `useTO`

```
useHS=find(HS>min(Time(show)) & HS<=max(Time(show)));
useTO=find(To>min(Time(show)) & To<=max(Time(show)));
```

9.2.1.1.2.4 Subplot 1

In the first subplot in the top left (`hh(1)`) a single second of data is plotted for the `Heel` and `Toe` data in the third dimension (i.e. movement in the Z/vertical axis). Heel marker movement is plotted in blue dots and heel movement in red.

```
plot(hh(1), Time(show), squeeze(MData(show,3,Heel)), '.b', ...
    Time(show), squeeze(MData(show,3,Toe)), '.r');
axis(hh(1), 'tight'); hold(hh(1), 'on'); yy=ylim(hh(1));
```

Any existing data is plotted with heelstrike (HS) in black ('k') and toe off (TO) in green ('g').

```
for j=1:length(useHS);plot(hh(1), HS(useHS(j))*[1 1], yy, 'k'); end
for j=1:length(useTO);plot(hh(1), TO(useTO(j))*[1 1], yy, 'g'); end
```

The subplot `hh(1)` is labelled.

```
ylabel('Height of heel (blue) and toe (red)')
```

9.2.1.1.2.5 Subplot 2

In the second subplot in the top middle (`hh(2)`) a single second of data is plotted for the differentiated `Heel` and `Toe` data in the first dimension (i.e. velocity in the x/horizontal axis). Heel marker velocity is plotted in blue dots and heel movement in red.

```

plot(hh(2), Time(show), [0; diff(squeeze(MData(show,1,Heel)))] , '. ', ...
    Time(show), [0; diff(squeeze(MData(show,1,Toe)))] , '.r');
axis(hh(2), 'tight');
ylim(hh(2), [-30 50]);
hold(hh(2), 'on');
yy=ylim(hh(2));

```

Any existing data is plotted with heelstrike (HS) in black ('k') and toe off (TO) in green ('g').

```

for j=1:length(useHS);plot(hh(2), HS(useHS(j))*[1 1], yy, 'k'); end
for j=1:length(useTO);plot(hh(2), TO(useTO(j))*[1 1], yy, 'g'); end

```

The subplot hh(2) is labelled.

```

ylabel('Forward speed of heel (blue) and toe (red)')

```

9.2.1.1.2.6 Subplot 3

In the third subplot in the top right (hh(3)) a single second of data is plotted for the differentiated Heel and Toe data in the third dimension (i.e. velocity in the z/vertical axis). Heel marker velocity is plotted in blue dots and heel movement in red.

```

plot(hh(3), Time(show), [0; diff(squeeze(MData(show,3,Heel)))] , '. ', ...
    Time(show), [0; diff(squeeze(MData(show,3,Toe)))] , '.r');
axis(hh(3), 'tight');
ylim(hh(3), [-10 30]);
hold(hh(3), 'on');
yy=ylim(hh(3));

```

Any existing data is plotted with heelstrike (HS) in black ('k') and toe off (TO) in green ('g').

```

for j=1:length(useHS);plot(hh(3), HS(useHS(j))*[1 1], yy, 'k'); end
for j=1:length(useTO);plot(hh(3), TO(useTO(j))*[1 1], yy, 'g'); end

```

The subplot hh(2) is labelled.

```

ylabel('Vertical speed of heel (blue) and toe (red)')

```

9.2.1.1.2.7 Subplot 4

In the fourth subplot on the bottom (hh(4)) the whole data set is plotted for the Heel and Toe data in the third dimension (i.e. movement in the z/vertical axis). Heel marker height is plotted in blue dots and heel height in red.

```
plot(hh(4), Time, squeeze(MData(:,3,Heel)), 'b', 'o', ...  
      Time, squeeze(MData(:,3,Toe)), 'r');  
ylim(hh(4),[-100 700]);  
hold(hh(4), 'on');  
yy=ylim(hh(4));
```

Any existing data is plotted with heelstrike (HS) in black ('k') and toe off (TO) in green ('g').

```
for j=1:length(HS);plot(hh(4), HS(j)*[1 1], yy, 'k'); end  
for j=1:length(TO);plot(hh(4), TO(j)*[1 1], yy, 'g'); end
```

9.2.1.1.2.8 Marking heel strike and Toe off

This section enables the user to mark heel strike (HS) and toe off (TO) on any of the above subplots using the fourth subplot (hh(4)) to arrange the other three (hh(1:3)) along the y axis i.e. for time. This marking is completed using a cross hair which is controlled by the mouse. The X coordinate of the cross hair is defined as X tick and the Y coordinate as Y tick.

First, the XTick and YTick are set as empty

```
set(hh(4), 'XTick', [], 'YTick', []);
```

The Title for the figure is determined from the matfile which calls the function and is placed above the first subplot (hh(1)).

```
title(hh(1), Title)
```

9.2.1.1.2.9 Ginput

The interaction with the mouse and keyboard is determined by the matlab function ginput. Within this function a right click, left click, mid click, left arrow, right arrow, and return key amongst other inputs can be assigned various functions.

In this programme the functions for `ginput(1)` are set as the following:

- Left click (`ginput` element 1) = mark foot strike
- Right click (`ginput` element 3) = mark toe off
- Mid click (`ginput` element 2) = erase nearest mark
- Left arrow (`ginput` element 29) = move left along the y axis
- Right arrow (`ginput` element 28) = move right along the y axis
- Return key = finish

```
[x , rub, but]=ginput(1);
if ~isempty(but);
    if but==1; HS=[HS x]; HS=sort(HS); end
    if but==3; TO=[TO x]; TO=sort(TO); end
    if but==2
        [ToD, ToNear]=min(abs(TO-x)); [HsD, HsNear]=min(abs(HS-x));
        if ToD<HsD; TO(ToNear)=[]; else HS(HsNear)=[]; end
    end

    if but==29; show=show+100; if max(show)>length(Time); show=show-100; end;
end
    if but==28; show=show-100; if min(show)<50; show=50:250; end; end
end
    end
```

Finally any data which runs off the end or beginning of the record is deleted and HS and TO times are displayed in the command window.

```
TO=sort(TO); HS=sort(HS);
TO(TO<min(HS))=[]; TO(TO>max(HS))=[];
dup=(find(diff(HS)<0.01)); HS(dup)=[];
dup=(find(diff(TO)<0.01)); TO(dup)=[];
disp(HS); disp(TO);
```

9.2.1.1.3 GetFileName Function

This function is used by several matlab scripts to identify the location of subject data through the use of a reference excel spreadsheet called `DATASET KEY.xlsx`.

9.2.1.1.3.1 Function Inputs and Outputs

The outputs from this function are as follows:

- Kinematic Data location (`CodaFile`)
- Subject name from `DATASET KEY.xlsx` (`Subj`)

- Designation of ipsilateral (i.e. previously injured) and contralateral (i.e. uninjured) limbs from DATASET KEY.xlsx (LR)
- Electromyographic data location (EmgFile)

The inputs to this function are as follows:

- Subject number (S)
- Speed of treadmill during data collection (Speed)

This data is summarised as follows

```
function [CodaFile, Subj, LR, EmgFile]= GetFileName(S, Speed)
```

The first section is a switch which indicates the sheet in DATASET KEY.xlsx ('Rel' or 'Sheet 1') from which to read data and the location of the data (Root). In the case of reliability data the matlab script which calls this function will do so by ensuring that the Speed = '' (i.e. is empty). Otherwise the data will be called by indicating Speed = 10, 15 or 20.

```
if isempty(Speed)
    Sheet='Rel';
    Root='C:\Users\wew295\Dropbox\MSc Data Previous hamstring injury\Reliability Raw Data\';
    Speed='15';
    IsRel=true;
else
    Sheet='Sheet1';
    Root='C:\Subject Raw Data\';
    IsRel=false;
end
```

The second section identifies where in DATASET KEY.xlsx data relating to the outputs are located. It also identifies any files which are missing and displays this information in the Command window.

```
[~, str]=xlsread('DATASET KEY.xlsx',Sheet , 'A2:D100');
Subj=char(str(1));
LR=char(str(3));

if IsRel
    CodaFile=[Root Subj(1:end-1) '\' Subj '\CODA\' Speed 'kph.mat'];
    EmgFile='To be found';
else
    CodaFile=[Root Subj '\CODA\' Speed 'km.mat'];
```



```

EmgFile=[Root Subj '\EMG\MAT files\' Speed 'KM.mat'];
if isempty(dir(CodaFile))
    CodaFile=[Root Subj '\CODA\' Speed 'kph.mat'];
end
end
if isempty(dir(CodaFile))
    disp([' GetFileName.m cannot find ' CodaFile])
end
end

```

9.2.1.1.4 HamsSegReg Script

This Matlab script extracts, plots and saves the segment rotation data for each individual. Using heel strike (already marked) this data is divided according to stride and an average segment rotation is calculated for each joint/segment in three planes (coronal, sagittal and transverse). This data is appended to the original CodaFile as MeanIReg for the mean data based on the ipsilateral heel strike data and MeanCReg for the mean data based on the contralateral heel strike data,

This script calls the following functions:

- GetSegRots to extract segment rotation data ordered according to known joints/segments.
- saveppt2 for saving figures to powerpoint.

9.2.1.1.4.1 Section 1

Initially the workspace is cleared and the following elements are defined:

- Speed is set to the running speed for analysis
- SegName are defined as various segments/joints
- PPF is the naming of the output powerpoint according to speed
- PPO calls the saveppt2 function to create the powerpoint PPF

```

clear
Speed='20';
SegNames={'Pelvis' 'Hip' 'Knee' 'Ankle' 'Foot'};
PPF=['HamSegRotSubj_' Speed '.pptx'];
PPO=saveppt2(PPF,'init');

```

9.2.1.1.4.2 Section 2

This is a loop which calls the following data for all defined subjects (S) from the CodaFile and passes them to the GetSegRots function as outputs for compiling and ordering:

- ISegRots = segment rotation data on the ipsilateral (i.e. previously injured) side
- CSegRots = segment rotation data on the contralateral (i.e. uninjured) side

- IHS = heel strike time points on the ipsilateral (i.e. previously injured) side
- CHS = heel strike time points on the contralateral (i.e. uninjured) side
- ITO = toe off time points on the ipsilateral (i.e. previously injured) side
- CTO = toe off time points on the contralateral (i.e. uninjured) side

In addition the following variables are defined:

- Time = stored time (hz) converted to seconds (i.e. divided by 200).
- IHSx = Ipsilateral heel strike time points (IHS) rounded to the nearest decimal
- CHSx = Contralateral heel strike time points (CHS) rounded to the nearest decimal
- Label = Subject code and speed data

```
for S=1:17;
[~, ISegRots CSegRots IHS CHS ITO CTO Subj File]=GetSegRots(Speed,S);
Time=(0:length(ISegRots)-1)/200;
IHSx=round(IHS*200); CHSx=round(CHS*200);
Label=[Subj Speed];
```

9.2.1.1.4.3 Section 3

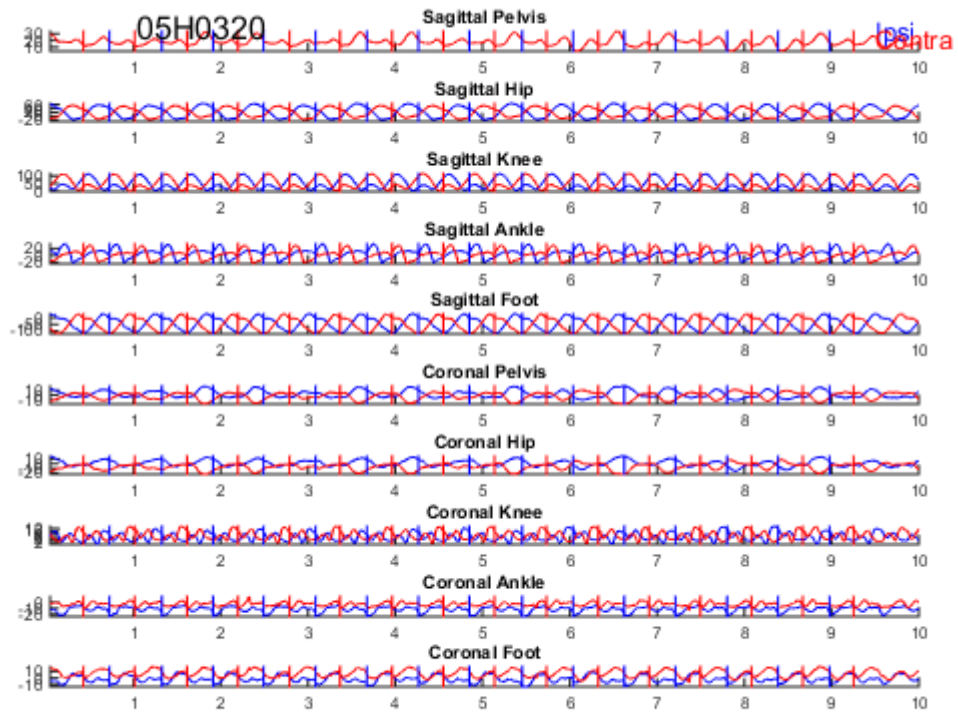
This section creates a figure of the segment rotations in the sagittal and coronal planes with heel strike data (IHS CHS) for checking. The first 10 data points are omitted. The output is a powerpoint file and figure with ipsilateral data in blue and contralateral data in red and labelled accordingly.

```
figure(1); clf;
for j=1:10; hh(j)=subplot(10,1,j); set(hh(j), 'FontSize', 6); hold(hh(j), 'on');
end
text(0.1, 1.2, Label, 'Units', 'normalized', 'Parent', hh(1), 'FontSize', 12);
text(0.95, 0.9, 'Ipsi', 'Units', 'normalized', 'Parent', hh(1), 'FontSize', 10,
'color', 'b');
text(0.95, 0.5, 'Contra', 'Units', 'normalized', 'Parent', hh(1), 'FontSize', 10,
'color', 'r');
for chan=1:5;
use=10:length(Time);
plot(hh(chan), Time(use), ISegRots(use,2,chan), 'b', Time(use),
CSegRots(use,2,chan), 'r');
axis(hh(chan), 'tight'); title(hh(chan),['Sagittal ' SegNames{chan}])
plot(hh(chan+5), Time(use), ISegRots(use,1,chan), 'b', Time(use),
CSegRots(use,1,chan), 'r');
axis(hh(chan+5), 'tight'); title(hh(chan+5),['Coronal ' SegNames{chan}])
for j=1:length(IHSx)
plot(hh(chan), [1 1]*IHS(j), ylim(hh(chan)), 'b')
plot(hh(chan+5),[1 1]*IHS(j), ylim(hh(chan+5)), 'b')
end
for j=1:length(CHSx)
```

```

        plot(hh(chan), [1 1]*CHS(j), ylim(hh(chan)), 'r')
        plot(hh(chan+5),[1 1]*CHS(j), ylim(hh(chan+5)), 'r')
    end
end
saveppt2('ppt', PPO, 'd', 'meta', 'res', 500)

```



9.2.1.1.4.4 Section 4

In this section the segment data is divided up and average according to strides.

Firstly the rounded heel strikes (HSx) and segment rotation data (SegRots) is defined as ipsilateral or contralateral.

```

for IC=1:2
    if IC==1; HSx=IHSx; SegRots=ISegRots;
    else HSx=CHSx; SegRots=CSegRots; end
end

```

Next, a variable called Reg is created. This is the registered (i.e. divided by stride) segment rotation data according to heel strike.

Reg is cleared

```
clear Reg
```

The divided segment data are stacked using the following variables within the loop below:

- j = the number of heel strikes HSx .
- ST = the stride time i.e. the number of time points between heel strikes defined as

$$HSx(j+1) - HSx(j)$$

```
for j=1:length(HSx)-1
```

```
    ST=HSx(j+1)-HSx(j);
```

Any strides which run off the beginning or end of the record are omitted

```
if HSx(j)>130 && HSx(j)<length(SegRots)-130
```

Next each stride is interpolated over 101 points (q) and defined as use.

```
for q=1:101
```

```
    Start=HSx(j)-ST/2; width=ST/100;
```

```
    use=round((Start+(q-1)*width):(Start+ q*width));
```

Next Reg is defined as the mean of SegRots for all interpolated strides (use) across all time points and dimensions.

```
Reg(q,1:3,1:5,j)=squeeze(mean(SegRots(use,:,:)));
```

```
end
```

```
end
```

```
end
```

Finally, the averaging of Reg according to ipsilateral and contralateral (IC) produces the average segment rotations for the ipsilateral (MnSegRotIReg) and contralateral (MnSegRotCReg) sides

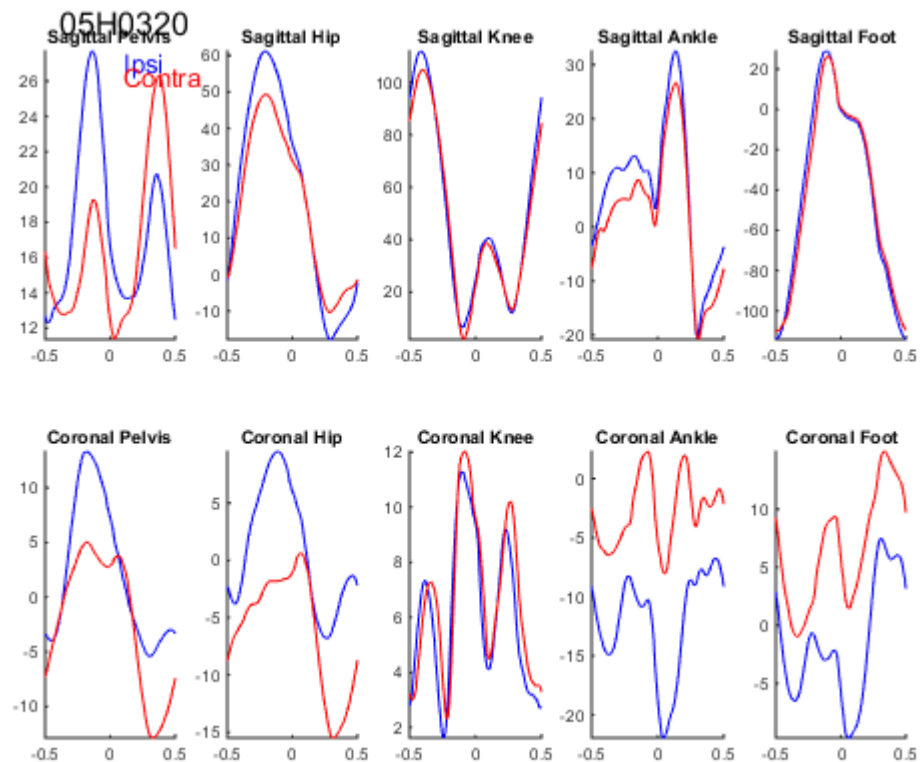
```
if IC==1; MnSegRotIReg=squeeze(nanmean(Reg,4)); else
```

```
MnSegRotCReg=squeeze(nanmean(Reg,4));end end
```

9.2.1.1.4.5 Section 5

The average segment rotations in the sagittal and coronal planes for the ipsilateral (MnSegRotIReg) and contralateral (MnSegRotCReg) sides are plotted in blue and red for each subject and labelled accordingly for checking.

```
figure(2); clf
for j=1:10; hh(j)=subplot(2,5,j); set(hh(j), 'FontSize', 6); hold(hh(j), 'on');
end
text(0.1, 1.1, Label, 'Units', 'normalized', 'Parent', hh(1), 'FontSize', 12);
text(0.6, 0.95, 'Ipsi', 'Units', 'normalized', 'Parent', hh(1), 'FontSize', 10,
'Color', 'b');
text(0.6, 0.9, 'Contra', 'Units', 'normalized', 'Parent', hh(1), 'FontSize', 10,
'Color', 'r');
for chan=1:5
c=chan;
yyI=squeeze(MnSegRotIReg(:,2,chan)); yyC=squeeze(MnSegRotCReg(:,2,chan));
plot(hh(c), -0.5:0.01:0.5, yyI, 'b', -0.5:0.01:0.5, yyC, 'r')
axis(hh(c), 'tight'); title(hh(c),['Sagittal ' SegNames{chan}])
yyI=squeeze(MnSegRotIReg(:,1,chan)); yyC=squeeze(MnSegRotCReg(:,1,chan));
plot(hh(c+5), -0.5:0.01:0.5, yyI, 'b', -0.5:0.01:0.5, yyC, 'r')
axis(hh(c+5), 'tight'); title(hh(c+5),['Coronal ' SegNames{chan}])
end
```



9.2.1.1.4.6 Section 6

The powerpoint images are saved and the average segment rotations for the ipsilateral (MnSegRotIReg) and contralateral (MnSegRotCReg) sides are appended to the CodaFile for each subject.

```
saveppt2('ppt', PPO, 'd', 'meta', 'res', 500)
save(File, 'MnSegRotIReg', 'MnSegRotCReg', '-append')
end
saveppt2(PPF, 'ppt', PPO, 'close');
```

9.2.1.1.4.7 GetSegRots.m

This function arranges the segment rotation data in each file according to the appropriate order, considering which leg is marked as the ipsilateral side.

Data relating to speed and subject identifiers (S) is passed to the function

```
function [Time ISegRots CSegRots IHS CHS ITO CTO Subj CodaFile]=GetSegRots(Speed,S)
%#ok<STOUT>

[CodaFile Subj LR]= GetFileName(S, Speed);
```

Error using GetSegRots (line 8)
Not enough input arguments.

All data relating to segment rotations are opened

```
load(CodaFile, 'SegRotData', 'SegRotNames', 'MarkerOcc', 'MarkerTime', 'IHS', 'ITO',
'CHS', 'CTO');
Time=MarkerTime(:,1); %#ok<NODEF>
size(SegRotData) %#ok<NODEF>
```

Depending on the designated side of interest, the segment rotations are stacked in Ipsilateral (ISegRots) or Contralateral (CSegRots) order.

```
if LR=='L'
    ISegRots=SegRotData(:,,[1 3 4 5 6]);
    CSegRots=SegRotData(:,,[2 7 8 9 10]);
else
    ISegRots=SegRotData(:,,[2 7 8 9 10]);
    CSegRots=SegRotData(:,,[1 3 4 5 6]);
end
```

9.2.1.1.5 HamsSegRU

This program collates the Segment rotation data according to previously injured and uninjured groups

```
clear
Speed='20'; % CHANGE SPEED HERE
SegNames={'Pelvis' 'Hip' 'Knee' 'Ankle' 'Foot'};
PPF=['SegRU_' Speed '.pptx'];
PPO=saveppt2(PPF,'init');
```

Extract mean segment rotation data for each subject in the previously injured group (H).

Concatenate the data into a single cell array of strings for each side:

- IRegHeapH (ipsilateral)
- CRegHeapH (contralateral)

```
IRegHeapH=[]; CRegHeapH=[];IRegHeapC=[]; CRegHeapC=[];
for H=1:9
    [MnSegRotIReg MnSegRotCReg Subj]= GetSegRegs(H, Speed);
    IRegHeapH=cat(4,IRegHeapH, MnSegRotIReg);
    CRegHeapH=cat(4,CRegHeapH, MnSegRotCReg);
end

% Calculate the group mean and standard error for each limb

IRegMeanH=nanmean(IRegHeapH,4);
IRegSemH=nanstd(IRegHeapH,[],4)/sqrt(9);
CRegMeanH=nanmean(CRegHeapH,4);
CRegSemH=nanstd(CRegHeapH,[],4)/sqrt(9);

% Calculate the mean difference between limbs and the standard error

RegMeanDiffH=nanmean(IRegHeapH-CRegHeapH,4);
RegSedH=nanstd(IRegHeapH-CRegHeapH,[],4)/sqrt(9);
```

Extract mean segment rotation data for each subject in the control group (C). Concatenate the data into a single cell array of strings for each side:

- IRegHeapC (ipsilateral)
- CRegHeapC (contralateral)

```
for C=10:17
    [MnSegRotIReg MnSegRotCReg Subj]= GetSegRegs(C, Speed);
    IRegHeapC=cat(4,IRegHeapC, MnSegRotIReg);
    CRegHeapC=cat(4,CRegHeapC, MnSegRotCReg);
```

```
end
```

```
% Calculate the group mean and standard error for each limb
```

```
IRegMeanC=nanmean(IRegHeapC,4);  
IRegSemC=nanstd(IRegHeapC,[],4)/sqrt(8);  
CRegMeanC=nanmean(CRegHeapC,4);  
CRegSemC=nanstd(CRegHeapC,[],4)/sqrt(8);
```

```
% Calculate the mean difference between limbs and the standard error
```

```
RegMeanDiffC=nanmean(IRegHeapC-CRegHeapC,4);  
RegSedC=nanstd(IRegHeapC-CRegHeapC,[],4)/sqrt(8);
```

Plot the results: set up figure 4 with subplots for each joint and plane

```
figure(4); clf  
for j=1:15; h4(j)=subplot(3,5,j); set(h4(j), 'FontSize', 6); hold(h4(j), 'on');  
end  
text(0.1, 1.15, 'All four legs', 'Units', 'normalized', 'Parent', h4(1),  
'FontSize', 12);  
text(0.4, 0.95, 'HamsIpsi', 'Units', 'normalized', 'Parent', h4(1), 'FontSize',  
10, 'Color', 'r');  
text(0.4, 0.90, 'HamsContra', 'Units', 'normalized', 'Parent', h4(1), 'FontSize',  
10, 'Color', 'g');  
text(0.4, 0.85, 'ConIpsi', 'Units', 'normalized', 'Parent', h4(1), 'FontSize',  
10, 'Color', 'k');  
text(0.4, 0.80, 'ConContra', 'Units', 'normalized', 'Parent', h4(1), 'FontSize',  
10, 'Color', 'b');  
  
% set up figures 1 - 3 with subplots for each joint and plane  
  
tt=-0.5:0.01:0.5; Labels={'IpsiReg' 'ContraReg' 'ICDiff'};  
for IC=1:3  
    % Figure 1 is the ipsilateral limb data for both groups  
    if IC==1;  
        MnC=IRegMeanC; SemC=IRegSemC; MnH=IRegMeanH; SemH=IRegSemH;  
        % Figure 2 is the contralateral limb data for both groups  
    elseif IC==2  
        MnC=CRegMeanC; SemC=CRegSemC; MnH=CRegMeanH; SemH=CRegSemH;  
        % Figure 3 is the limb difference data for both groups  
    else  
        MnC=RegMeanDiffC; SemC=RegSedC; MnH=RegMeanDiffH; SemH=RegSedH;  
    end  
    % Draw the figures  
    figure(IC); clf  
    for j=1:15; hh(j)=subplot(3,5,j); set(hh(j), 'FontSize', 6); hold(hh(j), 'on');  
end  
    text(0.1, 1.15, Labels{IC}, 'Units', 'normalized', 'Parent', hh(1), 'FontSize',  
12);  
    text(0.4, 0.95, 'Cons', 'Units', 'normalized', 'Parent', hh(1), 'FontSize', 10,  
'Color', 'k');
```

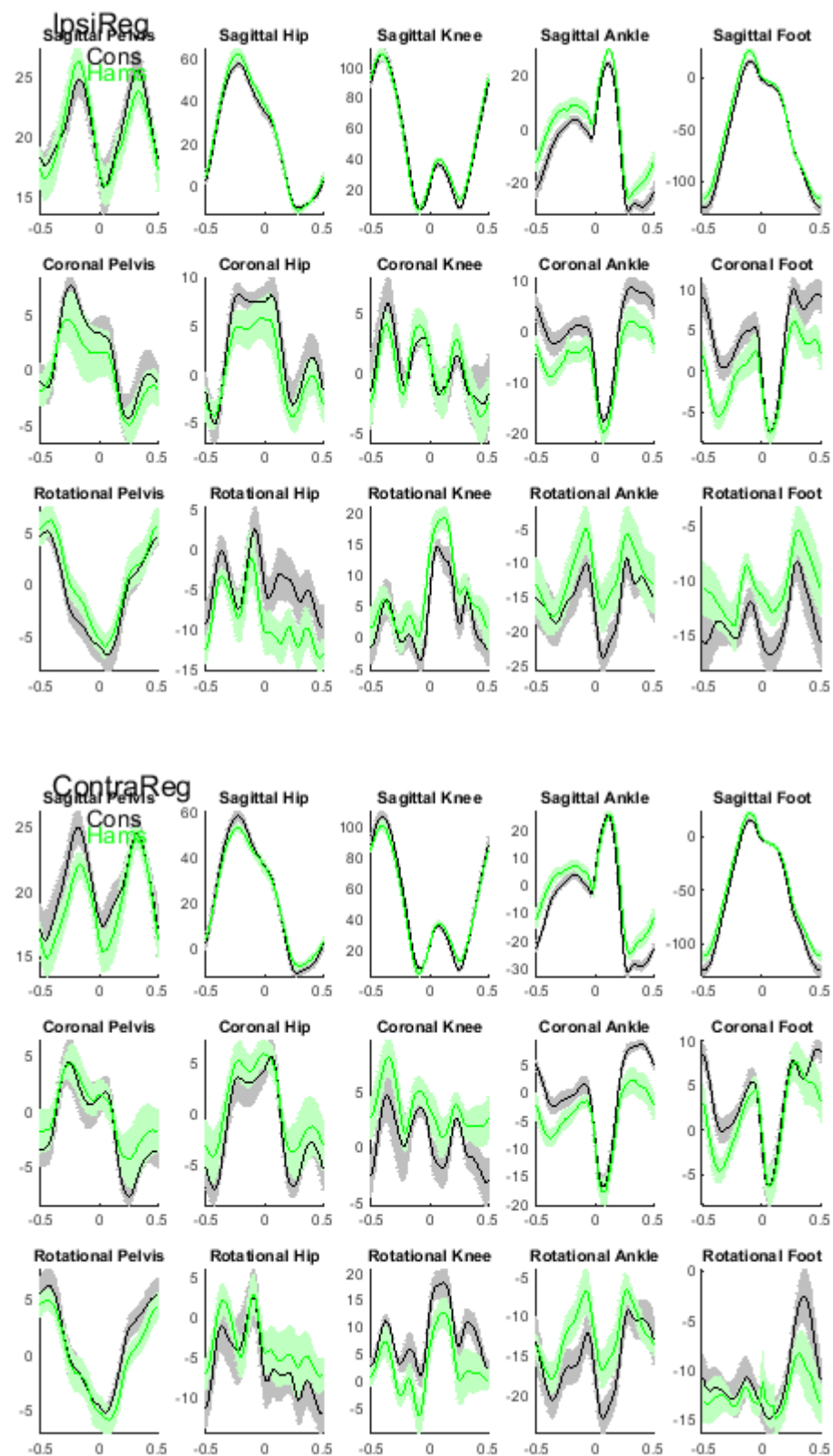


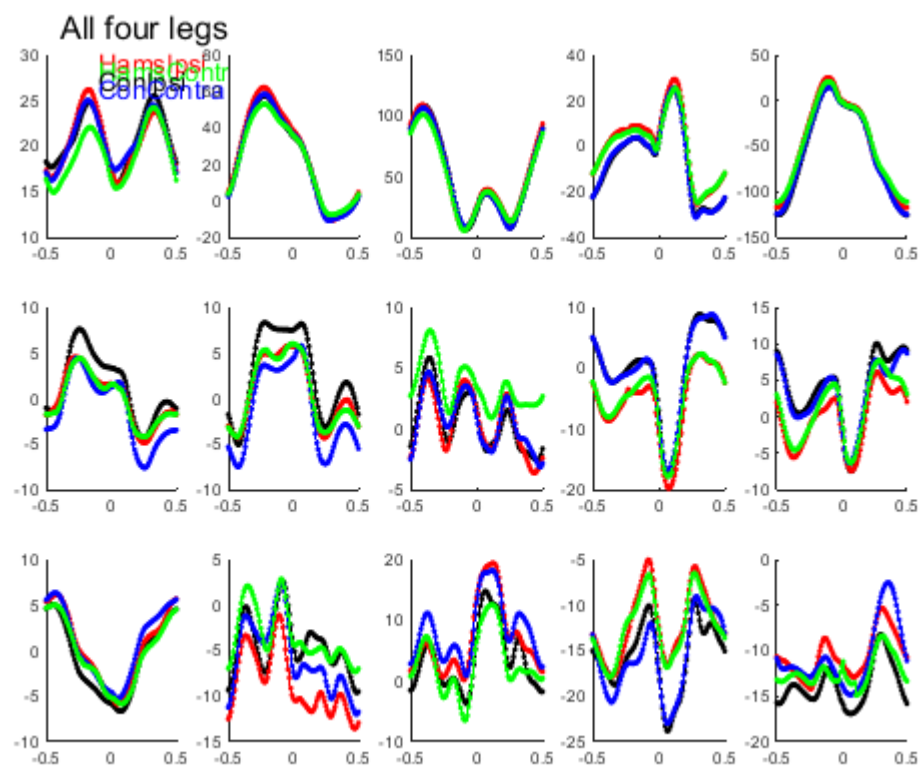
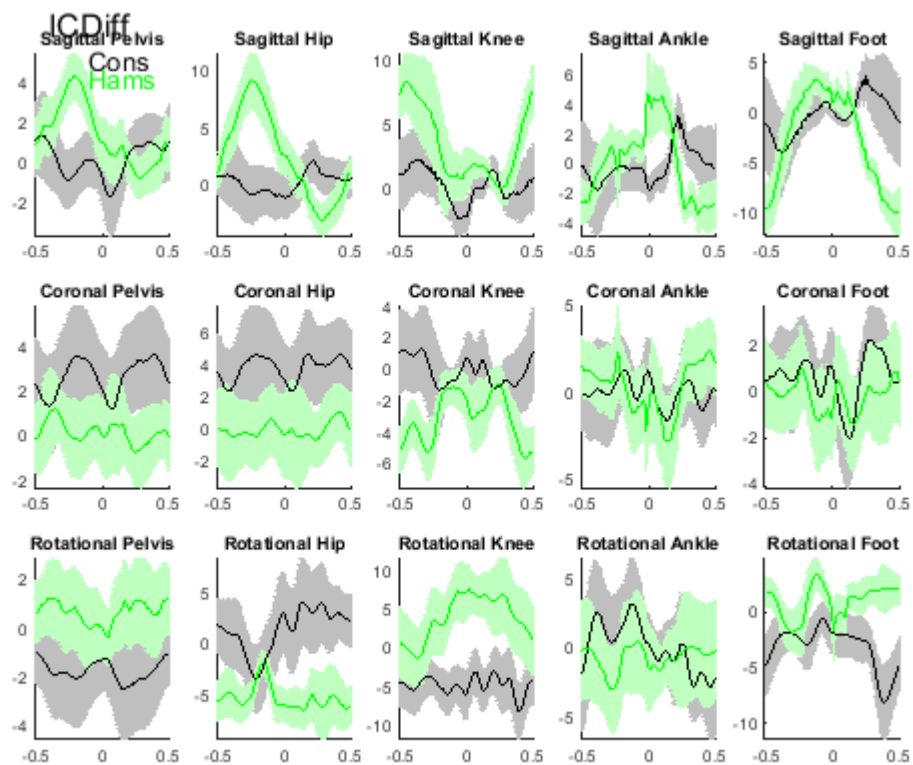
```

text(0.4, 0.85, 'Hams', 'Units', 'normalized', 'Parent', hh(1), 'FontSize', 10,
'color', 'g');
cols={'.-k','.-r';'.-b' '.-g'};
for chan=1:5;
    c=chan;
    yyC=MnC(:,2,chan); yyH=MnH(:,2,chan);
    eeC=SemC(:,2,chan); eeH=SemH(:,2,chan);
    errorbar(hh(c), tt,yyC,eeC,'color', [0.75 0.75 0.75])
    errorbar(hh(c), tt,yyH,eeH,'color', [0.75 1 0.75])
    plot(hh(c), tt,yyC, 'k', tt,yyH, 'g')
    if IC<3; plot(h4(c), tt, yyC, cols{IC,1}, tt, yyH, cols{IC,2}); end
    axis(hh(c), 'tight'); title(hh(c),['Sagittal ' SegNames{chan}])
    c=chan+5;
    yyC=MnC(:,1,chan); yyH=MnH(:,1,chan);
    eeC=SemC(:,1,chan); eeH=SemH(:,1,chan);
    errorbar(hh(c), tt,yyC,eeC,'color', [0.75 0.75 0.75])
    errorbar(hh(c), tt,yyH,eeH,'color', [0.75 1 0.75])
    plot(hh(c), tt,yyC, 'k', tt,yyH, 'g')
    if IC<3; plot(h4(c), tt, yyC, cols{IC,1}, tt, yyH, cols{IC,2}); end
    axis(hh(c), 'tight'); title(hh(c),['Coronal ' SegNames{chan}])
    c=chan+10;
    yyC=MnC(:,3,chan); yyH=MnH(:,3,chan);
    eeC=SemC(:,3,chan); eeH=SemH(:,3,chan);
    errorbar(hh(c), tt,yyC,eeC,'color', [0.75 0.75 0.75])
    errorbar(hh(c), tt,yyH,eeH,'color', [0.75 1 0.75])
    plot(hh(c), tt,yyC, 'k', tt,yyH, 'g')
    if IC<3; plot(h4(c), tt, yyC, cols{IC,1}, tt, yyH, cols{IC,2}); end
    axis(hh(c), 'tight'); title(hh(c),['Rotational ' SegNames{chan}])
    ysc(chan,:)=ylim(hh(c));
end

end

```





Save figures in powerpoint

```
for j=1:3;
    figure(j);
    saveppt2('ppt', PPO, 'd', 'meta', 'res', 500);
end
saveppt2(PPF, 'ppt', PPO, 'close');
```

9.2.1.1.6 BootstrapArea

This program executes a bootstrap comparison of the 'real' dataset to 1 million comparative datasets. Areas of the data when contiguous records of difference between groups are identified. The effect size is calculated and contiguous records where this is greater than 1 is identified. Both the length and magnitude of this difference is measured. The bootstrap randomised the time order of the original datasets and the frequency of effect sizes of the same magnitude is calculated. In this way the time order of the original dataset is respected.

```
clear
load HamsSegRU.mat IRegHeapC IRegHeapH CRegHeapC CRegHeapH
for s=1:5
    for p=1:3
        CRec=squeeze(IRegHeapC(:,p,s,:)-CRegHeapC(:,p,s,:));
        HRec=squeeze(IRegHeapH(:,p,s,:)-CRegHeapH(:,p,s,:));
        Rec=[CRec HRec];
```

9.2.1.1.6.1 Look first at the real data

Create group A

```
nA=1:8; %this is the C group
A=Rec(:,nA);
MnA=mean(A,2);
SeA=std(A,[],2)/sqrt(9);

% Create group B
nB=9:17; %this is the H group
B=Rec(:,nB);
MnB=mean(B,2);
SeB=std(B,[],2)/sqrt(8);
```

Calculate the standard error of the difference (SED) and effect size (z =the mean difference/SED) Measure the length and magnitude of contiguous runs of record where $z > 1$

```

for j=1:2
    SED=sqrt(SeA.^2+SeB.^2);
    Z=(MnB-MnA)./SED;
    % find max run length of different effect sizes
    if j==2; Z=-1*Z; end
    IsUp=Z>1;
    Mx=0; Acc=0; MxA=0;
    for i=1:100
        if IsUp(i)
            Acc=Acc+Z(i);
            if Acc>Mx; Mx=Acc; end
            if Z(i)>MxA; MxA=Z(i); end
        else
            Acc=0;
        end
    end

    if j==1;
        MxE=Mx;
        MxAE=MxA;
    else
        MxE=max([MxE Mx]);
        MxAE=max([MxAE MxA]);
    end
end
end

```

9.2.1.1.6.2 Now do the bootstrap

```

N=1000000;
parfor k=1:N
    %
    disp(k);
    % Create group A
    nA=randi(17,9,1); % nA=1:8; %this is the C group
    A=Rec(:,nA);
    MnA=mean(A,2);
    SeA=std(A,[],2)/sqrt(9);

    % Create group B
    nB=randi(17,8,1); % nB=9:17; %this is the H group
    B=Rec(:,nB);
    MnB=mean(B,2);
    SeB=std(B,[],2)/sqrt(8);

    SED=sqrt(SeA.^2+SeB.^2);
    Z=(MnB-MnA)./SED;
    % find max run length of different effect sizes

    IsUp=Z>1;

```

```

        Mx=0; Acc=0; MxA=0;
        for i=1:100
            if IsUp(i)
                Acc=Acc+Z(i);
                if Acc>Mx; Mx=Acc; end
                if Z(i)>MxA; MxA=Z(i); end
            else
                Acc=0;
            end
        end
        AllMxs(k)=Mx;
        AllMxAs(k)=MxA;
    end
end

```

9.2.1.1.6.3 Compare real and bootstrap

Explore the probability that the length and magnitude of an effect size of >1 seen in the original data will appear in the randomised dataset

```

% assess probability of as big effects
Pr=2*sum(AllMxs>=MxE & AllMxAs>=MxAE)/N;
if Pr>1; Pr=1; end
Prob(s,p)=Pr;

disp([int2str([s p]) ' ' num2str(Prob(s,p))]);

end
end

save BootstrapArea.mat MxE MxAE Prob AllMxs AllMxAs N

```

9.2.1.2 EMG

9.2.1.2.1 HamEMGReg

This program divides the EMG record according to strides and calculates a subject mean for each EMG channel and EMG ratio.

```

clear
Speed='20'; % CHANGE SPEED HERE
%Ratios calculated are from 1:2 to 1:10 i.e. 9 ratios
LabNames={'Biceps femoris', 'I glut max', 'I ES', 'I Rectus Fem', 'I Ext
Oblique',...
          'C glut max', 'C ES', 'C Rectus Fem', 'C Ext Oblique', 'Sum'};
PPF=['EMGReg_' Speed '.ptx'];
PPO=saveppt2(PPF,'init');

```

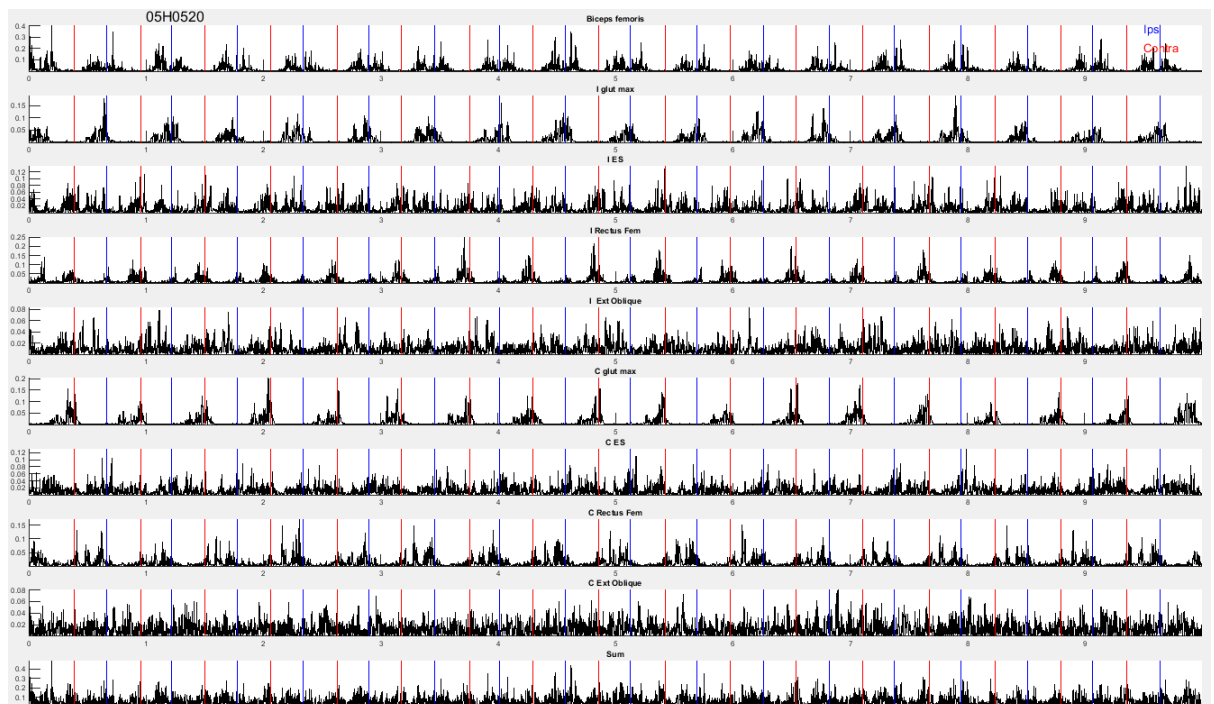
A loop selects each subject file (s) and uses the GetEMGs and GetHeelToe functions to extract the EMG and stride time data. The heel strike (and toe off) time are rounded and multiplied by 1200 to match the EMG time record (1200Hz).

```
for S=1:18; % CHANGE THE SUBJECT HERE

% get the EMG data & heel strike times
[EMGs , ~, Time]= GetEMGs(S, Speed);
Time=Time(1:length(EMGs)); % some Time records are 1 point too long!
EMGs(:,10)=sum(EMGs,2); %add a 10th channels with the total emg value
[t a b c d IHS CHS e f Subj File ]=GetHeelToe(Speed,S);
IHSx=round(IHS*1200); CHSx=round(CHS*1200);
Label=[Subj Speed];
```

The layout is defined for the first figure which allows inspection of the EMG record and heel strikes across the entire record

```
figure(1); clf;
for j=1:10; hh(j)=subplot(10,1,j); set(hh(j), 'FontSize', 6); hold(hh(j), 'on');
end
text(0.1, 1.2, Label, 'Units', 'normalized', 'Parent', hh(1), 'FontSize', 12);
text(0.95, 0.9, 'Ipsi', 'Units', 'normalized', 'Parent', hh(1), 'FontSize', 10,
'color', 'b');
text(0.95, 0.5, 'Contra', 'Units', 'normalized', 'Parent', hh(1), 'FontSize', 10,
'color', 'r');
```



This loop rectifies the EMG, subtracts the mean to deal with any offsets and plots EMGs with HS marks for inspection

```
for chan=1:10;
    EMGs(:,chan)=abs(EMGs(:,chan)-mean(EMGs(:,chan)));
    plot(hh(chan), Time, EMGs(:,chan), 'k');
    axis(hh(chan), 'tight'); title(hh(chan),LabNames{chan})
    for j=1:length(IHSx)
        plot(hh(chan),[1 1]*IHS(j), ylim(hh(chan)), 'b')
    end
    for j=1:length(CHStx)
        plot(hh(chan),[1 1]*CHSt(j), ylim(hh(chan)), 'r')
    end
end
savepvt2('pvt', PPO, 'd', 'meta', 'res', 500)
```

This loop cuts out and stacks registered time slices of EMG for each channel and ratio. Mean EMG data is log transformed and interpolated across 101 points. Data for the ipsilateral stride and contralateral stride is calculated and saved as MeanIReg and MeanCReg.

```
for IC=1:2
    if IC==1; HSx=IHSx; else HSx=CHStx; end
    clear Reg
    for j=1:length(HSx)-1
        ST=HSx(j+1)-HSx(j); % this is a number of EMG points between heel strikes
        if HSx(j)>800 && HSx(j)<length(EMGs)-800 % ignore places that would run
off the end or beginning of record
            for q=1:101
                % each point in bits is the mean of n points of rectified raw
emg.

                Start=HSx(j)-ST/2; width=ST/100;
                use=round((Start+(q-1)*width):(Start+ q*width));
                Reg(q,1:10,j)=log(mean(EMGs(use, :))); %#ok<*SAGROW>
                Reg(q,11:19,j)=Reg(q,1,j)-Reg(q,2:10,j); % add the ratios
            end
        end
    end

    if IC==1; MeanIReg=squeeze(nanmean(Reg,3)); else
MeanCReg=squeeze(nanmean(Reg,3)); end% averaging across strides
end
```

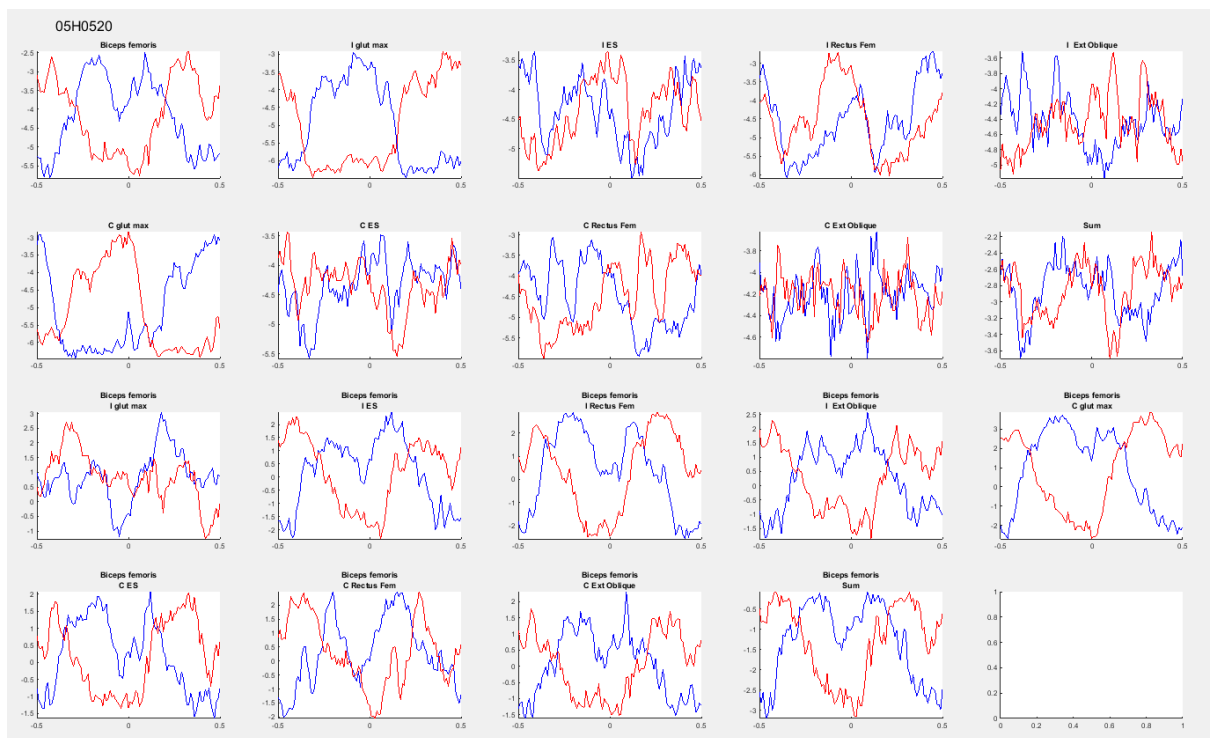
The registered EMG data is plotted for inspection.


```

figure(2); clf
for j=1:20; hh(j)=subplot(4,5,j); set(hh(j), 'FontSize', 6); hold(hh(j), 'on');
end
text(0.1, 1.2, Label, 'Units', 'normalized', 'Parent', hh(1), 'FontSize', 12);

for chan=1:19;
yy=squeeze(MeanIReg(:,chan));
plot(hh(chan), -0.5:0.01:0.5, yy, 'b')
yy=squeeze(MeanCReg(:,chan));
plot(hh(chan), -0.5:0.01:0.5, yy, 'r')
if chan<11;
title(hh(chan),LabNames{chan})
else
title(hh(chan),{LabNames{1} LabNames{chan-9}})
end
axis(hh(chan), 'tight');
end
end

```



MeanIReg and MeanCReg are saved and the figures are exported to a powerpoint

```

saveppt2('ppt', PPO, 'd', 'meta', 'res', 500)
save(File, 'MeanIReg', 'MeanCReg', '-append')

end
saveppt2(PPF, 'ppt', PPO, 'close');

```

9.2.1.2.2 GetEMGs

This function extracts and arranges the EMG records

The function uses the GetFileName function to locate the EMG data and define the ipsilateral and contralateral limbs.

```
function [EMGs, Subj, Time]= GetEMGs(S, Speed)

[CodaFile, Subj, LR, EmgFile]= GetFileName(S, Speed);

% The EMG channels are arranged into a standardised order depending on
% which side is ipsilateral

LOrder=[1 3 7 9 13 4 8 10 14 2];
ROrder=[2 4 8 10 14 3 7 9 13 1];

CL='R'; if LR==CL; CL='L'; end
if LR=='R'; Order=ROrder; else Order=LOrder; end
```

The channel names are identified to enable matching

```
if S<18

    CNames={'Biceps femoris', [LR ' glut max'], [LR ' ES'], [LR ' Rectus Fem'], [LR '
Ext Oblique'],...
           [CL ' glut max'], [CL ' ES'], [CL ' Rectus Fem'], [CL '
Ext Oblique'], 'Sum'};
    if LR=='L'; CNames{5}='L Ext Oblique';CNames{9}='R Ext Oblique'; end
```

The EMG data is extracted and channels identified according to name. Time in seconds is calculated by dividing by 1200 (data collected at 1200 Hz)

```
data=[];
load(EmgFile, 'data', 'labels'); %Time scale is 10 sec at 1200/s
Time=(0:length(data)-1)/1200;
CNos=[];
for k=1:length(CNames)
    CNos=[CNos strmatch(CNames{k}, labels)]; %#ok<MATCH2,AGROW>
end

EMGs=data(2:end,CNos); % in EMG is the raw data with the 9 channels in standard
Ord

end
```

9.2.1.2.3 HamsEMGRU

This programme collates the mean EMG data and ratios for each subject according to group and collates them into a group cell array of strings for analysis.

Define data sources and powerpoint outputs

```
clear
Speed='20'; % CHANGE SPEED HERE
Ratios=[1 2; 1 3; 1 4; 1 5; 1 6; 1 7; 1 8; 1 9; 1 10; 2 5; 2 9; 2 7; 2 3; 3 5; 3 9; 7
5; 7 9];
LabNames={'Biceps femoris', 'I glut max', 'I ES', 'I Rectus Fem', 'I Ext
Oblique',...
          'C glut max', 'C ES', 'C Rectus Fem', 'C Ext Oblique',
'Sum'};
PPF=['EMGRU_' Speed '.pptx'];
PPO=saveppt2(PPF,'init');

IRegHeapH=[]; CRegHeapH=[]; IRegHeapC=[]; CRegHeapC=[];
```

Concatenate the ipsilateral and contralateral EMG data which is extracted for each subject in the previously injured group using the GetEMGRefs function

```
for H=1:9;

    [MeanIReg MeanCReg Subj]= GetEMGRefs(H, Speed);
    IRegHeapH=cat(3,IRegHeapH, MeanIReg);
    CRegHeapH=cat(3,CRegHeapH, MeanCReg);
end
```

Calculate group mean and standard error for the ipsilateral, contralateral and difference between sides.

```
IRegMeanH=nanmean(IRegHeapH,3);
IRegSemH=nanstd(IRegHeapH,[],3)/sqrt(9);
CRegMeanH=nanmean(CRegHeapH,3);
CRegSemH=nanstd(CRegHeapH,[],3)/sqrt(9);
ICDiffH=nanmean(IRegHeapH(:,[2 2 3 4 5],:)-CRegHeapH(:,[6 6 7 8 9],:),3);
ICSedH=nanstd(IRegHeapH(:,[2 2 3 4 5],:)-CRegHeapH(:,[6 6 7 8 9],:),[],3)/sqrt(9);
```

Repeat the process for the control group

```
for C=10:17;

    [MeanIReg MeanCReg Subj]= GetEMGRefs(C, Speed);
```

```

IRegHeapC=cat(3,IRegHeapC, MeanIReg);
CRegHeapC=cat(3,CRegHeapC, MeanCReg);
end
IRegMeanC=nanmean(IRegHeapC,3);
IRegSemC=nanstd(IRegHeapC,[],3)/sqrt(8);
CRegMeanC=nanmean(CRegHeapC,3);
CRegSemC=nanstd(CRegHeapC,[],3)/sqrt(8);
ICDiffC=nanmean(IRegHeapC(:,[2 2 3 4 5],:)-CRegHeapC(:,[6 6 7 8 9],:),3);
ICSedC=nanstd(IRegHeapC(:,[2 2 3 4 5],:)-CRegHeapC(:,[6 6 7 8 9],:),[],3)/sqrt(8);

```

Plot the group comparison data (Mean and SEM) for the ipsilateral side.

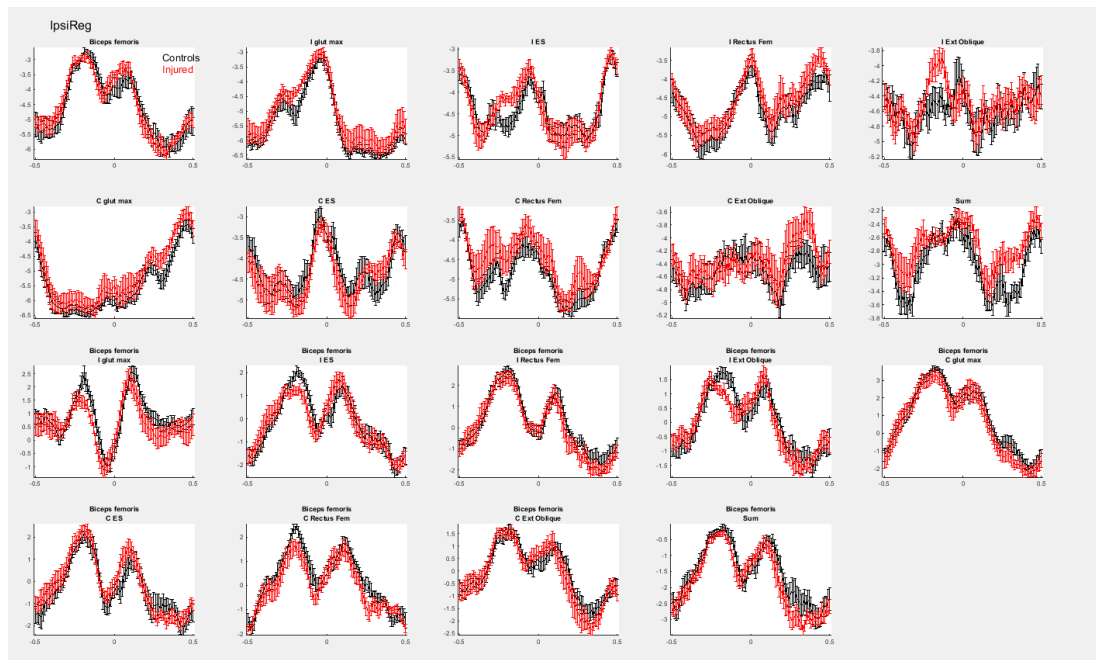
```

tt=-0.5:0.01:0.5; Labels={'IpsiReg' 'ContraReg' 'ICDiff'};
for IC=1
    if IC==1
        MnC=IRegMeanC; SemC=IRegSemC; MnH=IRegMeanH; SemH=IRegSemH;
    elseif IC==2
        MnC=CRegMeanC; SemC=CRegSemC; MnH=CRegMeanH; SemH=CRegSemH;
    else
        MnC=ICDiffC; SemC=ICSedC; MnH=ICDiffH; SemH=ICSedH;
    end
    figure(IC); clf
    for j=1:19; hh(j)=subplot(4,5,j); set(hh(j), 'FontSize', 6); hold(hh(j), 'on');
end
    text(0.1, 1.2, Labels{IC} , 'Units', 'normalized', 'Parent', hh(1), 'FontSize',
12);
    text(0.8, 0.9, 'Controls', 'Units', 'normalized', 'Parent', hh(1), 'FontSize',
10, 'Color', 'k');
    text(0.8, 0.8, 'Injured', 'Units', 'normalized', 'Parent', hh(1), 'FontSize', 10,
'Color', 'r');

    for chan=1:19;
        if IC==3 && (chan==1 || chan>5); continue; end
        yyC=MnC(:,chan); yyH=MnH(:,chan);
        eeC=SemC(:,chan); eeH=SemH(:,chan);
        errorbar(hh(chan), tt,yyC,eeC,'k')%'color', [0.75 0.75 0.75])
        errorbar(hh(chan), tt,yyH,eeH,'r')%'color', [0.75 1 0.75])
        plot(hh(chan), tt,yyC, 'k', tt,yyH, 'r')
        if chan<11;
            title(hh(chan),LabNames{chan})
        else
            title(hh(chan),{LabNames{1} LabNames{chan-9}})
        end
        axis(hh(chan), 'tight');
        yy=ylim(hh(chan));
        ysc(chan,:)=yy;
    end
    saveppt2('ppt', PPO, 'd', 'meta', 'res', 500)

end
saveppt2(PPF,'ppt',PPO,'close');

```



9.2.1.2.4 GetEMGRefs

This function extracts the mean EMG data for each subject using the GetFileName function

```
function [MeanIReg MeanCReg Subj]= GetEMGRefs(S, Speed)
[CodaFile Subj]=GetFileName(S,Speed);
MeanIReg=[]; MeanCReg=[];
load(CodaFile, 'MeanIReg', 'MeanCReg')
```

9.2.1.2.5 BootstrapEMG

This program performs a bootstrap analysis. Firstly any contiguous record where the standard error of the difference between the ratio data of test groups is greater than the difference between group means is measured in both length and magnitude. The bootstrap explores the number of instances where a difference of the same or greater magnitude exists in a sample of 1 million pairs of datasets constructed by randomising the time order of the original datasets.

The ratio data is first extracted using the HamsEMGRU program and collated into nA (control) and nB (previously injured) groups.

```

clear

load HamSEMGRU.mat IRegHeapC IRegHeapH;
for c=11:18

    CRec=squeeze(IRegHeapC(:,c,:));
    HRec=squeeze(IRegHeapH(:,c,:));
    Rec=[CRec HRec];

    nA=1:8; %this is the C group
    A=Rec(:,nA);
    MnA=mean(A,2);
    SeA=std(A,[],2)/sqrt(9);

    % Create group B
    nB=9:17; %this is the H group
    B=Rec(:,nB);
    MnB=mean(B,2);
    SeB=std(B,[],2)/sqrt(8);

```

The standard error of the difference is calculated and any parts of the record when the Mean difference between groups/SED is >1 identified and measured in both length and magnitude

```

for j=1:2
    SED=sqrt(SeA.^2+SeB.^2);
    Z=(MnB-MnA)./SED;
    % find max run length of different effect sizes
    if j==2; Z=-1*Z; end
    IsUp=Z>1;
    Mx=0; Acc=0; MxA=0;
    for i=1:50
        if IsUp(i)
            Acc=Acc+Z(i);
            if Acc>Mx; Mx=Acc; end
            if Z(i)>MxA; MxA=Z(i); end
        else
            Acc=0;
        end
    end

    if j==1;
        MxE=Mx;
        MxAE=MxA;
    else
        MxE=max([MxE Mx]);
        MxAE=max([MxAE MxA]);
    end
end
end

```

The bootstrap is then performed on 1 million comparative datasets which are formed by randomising the time order of the originals.

```

N=1000000;
parfor k=1:N

%      disp(k);
% Create group A
nA=randi(17,9,1); % nA=1:8; %this is the C group
A=Rec(:,nA);
MnA=mean(A,2);
SeA=std(A,[],2)/sqrt(9);

% Create group B
nB=randi(17,8,1); % nB=9:17; %this is the H group
B=Rec(:,nB);
MnB=mean(B,2);
SeB=std(B,[],2)/sqrt(8);

```

The same Mean difference/SED calculation is performed

```

SED=sqrt(SeA.^2+SeB.^2);
Z=(MnB-MnA)./SED;
% find max run length of different effect sizes

IsUp=Z>1;
Mx=0; Acc=0; MxA=0;
for i=1:50
    if IsUp(i)
        Acc=Acc+Z(i);
        if Acc>Mx; Mx=Acc; end
        if Z(i)>MxA; MxA=Z(i); end
    else
        Acc=0;
    end
end
AllMxs(k)=Mx;
AllMxAs(k)=MxA;

end

```

A comparison is made between real and bootstrap to assess probability of as big effect in the bootstrap data

```

Pr=2*sum(AllMxs>=MxE & AllMxAS>=MxAE)/N;
if Pr>1; Pr=1; end
Prob(c)=Pr;

disp([int2str(c) ' ' num2str(Prob(c))]);

```

```

11 0
12 0
13 1
14 0
15 0
16 1
17 0
18 1

```

```

end

save BootstrapEMG.mat MxE MxAE Prob AllMxs AllMxAS N

```


9.2.2 Nordic and other EMG array data (Chapter 6, and 7)

9.2.2.1 Overview

All data starts as a TMSI datafile

Nordic_Extract_Check_CD.m

Converts the TMSI data file to matlab struct file containing emg, force and trigger data

Plots figures to check quality of data

Stuct2rawemg_Nordic.m

Converts struct file to 64 channels of EMG in 'rawemg', force data to kgwt and trigger and saves to matfile

Filter_Check_Nordic_CD.m

Performs band pass (10-500hz) and notch (@50hz) filtering on EMG data. Saves to FiltDat

Plots FiltDat, Left and Right Force and Trigger data for inspection – any erroneous data is deleted manually

CHANNEL CONTRIBUTION TO OVERALL MAXIMAL ACTIVITY

GrEq_Nordic_CD.m

Calculates the contribution of each channel to total EMG activity in the limb during the maximal activation period across the limb. Saves this as ChMaxRel

NordicExcelExport_CD.m

Outputs ChMaxRel values for left and right limbs to an excel workbook with a new sheet for each trial/subject

PEAK RMSEMG (NORMALISED TO MEAN ACTIVITY) DURING FORCE GENERATION

NordicZero2Nan_CD.m

Replaces all zeros in FiltDat with Nans

Fq_ForceOn_Off_Nordic_CD.m

*Identifies force on and force off data using visual inspection and marking of force records and store to matfile using **MarkFqForce.m** function*

RMSEMG_Nordic_CD

Extracts Filtered EMG during the force time, split according to limb and calculate RMS EMG using a 50ms window, convert to mV from uV and store to matfile as LEMGMV and REMGMV

Nordic_NormRMSEMGCalc_CD

Calculates normalised to mean RMS values across each record

NordicPeakNormEMG_CD

Calculates peak RMSEMG per channel (normalised to mean RMSEMG)

NordicPeakNormExcel_CD

Exports peak RMSEMG values to an excel spreadsheet

iEMG DURING FORCE GENERATION

Interpolate_for_iEMG_Nordic_CD

Performs a resampling (i.e. interpolation) of data across a number of points equal to the longest RMSEMG record for each side across all trials to 'time normalise' the dataset

iEMG_Nordic_CD

Calculates the area under the interpolated RMSEMG curve using the trapz function (the iEMG value), divides the value by 1000, and saves as LiEMG and RiEMG in the matfile

iEMG_Nordic_Excel_CD

Exports the iEMG values to an excel spreadsheet

MEDIAN FREQUENCY DURING FORCE GENERATION

Interpolate_Nan_FiltDat_Nordic_MDF_CD

Prepares FiltDat for frequency analysis by interpolating across any Nans and dividing the data into left and right. Data is extracted during the force time for analysis and saved as useLFiltDat and useRFiltDat in the matfile

Nordic_MDF_CD

Calculates the frequency components of each signal during loading. Calculates the median frequency from the cumulative sum of the frequency outputs. Saves the median frequency value for each channel to the matfile as LMDF and RMDF

MDF_Nordic_Excel_CD

Outputs MDF data to an excel spreadsheet

PEAK FORCE

PeakForceExtract

Calculates the max force from the left and right force data and exports to an excel spreadsheet.

9.2.2.2 EMG related programs

9.2.2.2.1 Nordic_Extract_Check_CD

This script extracts the raw TMSI data for inspection using the TMSI_convert function provided by the manufacturer. Data are plotted on five graphs:

- Graph 1 and 2: EMG data for 32 channels left and right
- Graph 3 and 4: Force data left and right
- Graph 5: Trigger data

Script text

```
clear  
close all hidden
```

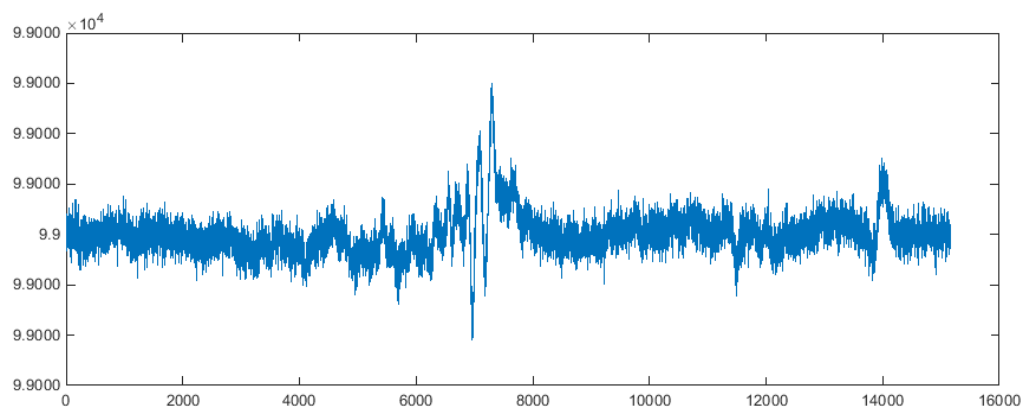
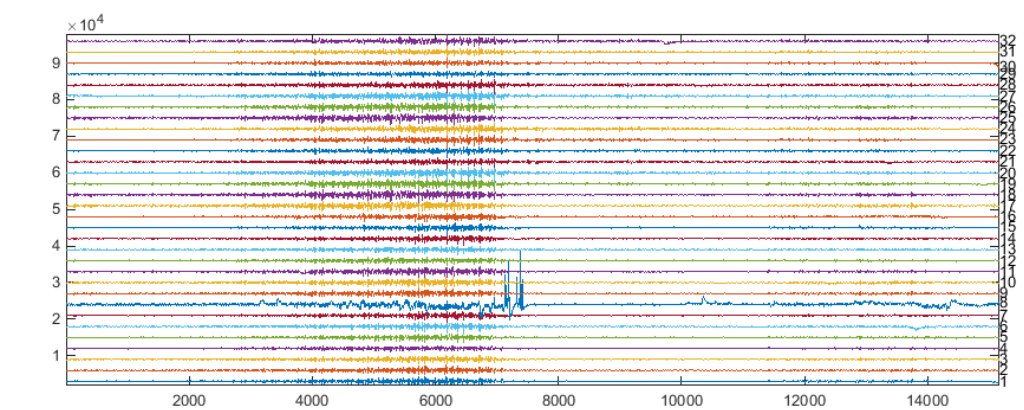
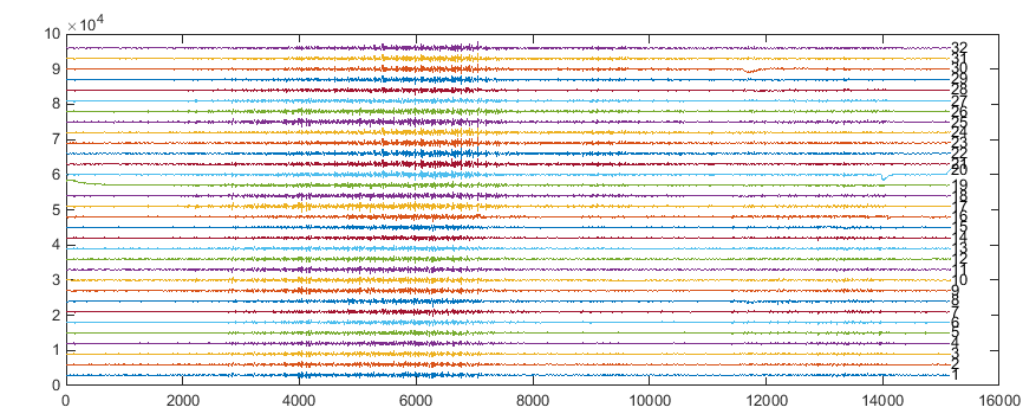
```

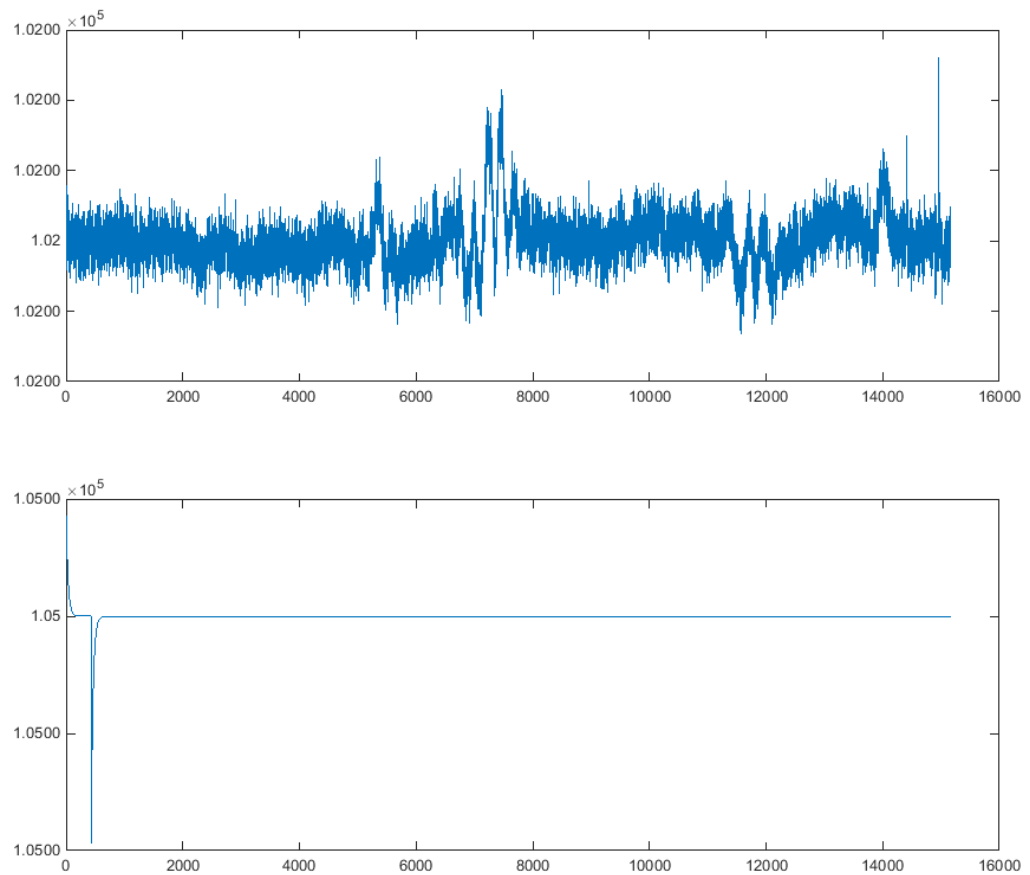
path='F:\PHD DATA\Study 2 - Nordic Activation\DATA\NOR14 -
Copy\NC01\EMG';%CHANGE_this is the location of the data
file='1.S0'; %CHANGE_this is the file identifier

for i=0:1
    filename=[file int2str(i)]; %this is the actual filename i.e. considers both TMSI
    file S.00 and S.01
    signal = tmsi_convert(path,filename); %this passes the S00 and S01 files to the
    tmsi_convert function for opening.
    signals(i+1)=signal;%this
    figure(i+1); set(gcf, 'windowstyle', 'docked'); clf %this sets a figure for each
    group of signals and the following script plots out the content of each EMG channel.
    for c=1:32% c equates to the first 32 channels i.e. the EMG channels
        yy=signal.data{c}(100:end);%yy is each EMG channel from 100 timepoints to the
        end (the first 100 timepoints are omitted as there are large negative values in the
        timeframe)
        plot(yy+c*3000)%each signal is plotted at 3000um positive offset to the next
        hold on %and each signal plot is held on the graph
        text(length(yy),yy(end)+c*3000,int2str(c)) %the channel number is added on
        each line
        drawnow
    end
end
axis tight
if i==1 %change this to 0 if the force/trigger signal is in S.00/1 if in S01
    (DEFAULT)
    for c=33:35;%these are the channel numbers for force and trigger signals
        figure(c); set(gcf, 'windowstyle', 'docked'); clf %the figure number matched
        the channel number
        yy=signal.data{c}(100:end);
        plot(yy+c*3000)
        if c==35;
            trigger=yy;
        end
        hold on
    end
end
end

MatFile='TMSIData_NC01.mat';%CHANGE_this saves the signal data as a matfile
save(MatFile, 'signals')

```





9.2.2.2.2 Struct2rawemg

This code extracts the EMG, force and trigger signals from the structure containing the data in the TSMI_converted matfile output.

In the first section the location of the file and the list of filenames is outlined. Parallel pooling of data processes is initiated to speed up the extraction process

```
Root='F:\PHD DATA\Study 2 - Nordic Activation\DATA\EXTRACTED EMG - Copy\Real\';
DS=dir([Root '*.mat']);
Files={DS.name};
% delete(gcp)
% parpool
```

In the second section the subfolders for EMG, left and right force and the trigger signals are created/cleared. The structure containing the data ('Signals') is opened for each file in a loop

```
for f=1:length(Files)
```

```
clear rawemg LForce RForce Trigger
File=Files{f};
load ([Root File], 'Signals')
```

In the third section the EMG data is extracted. The structure contains two subsections. Section 1 contains the EMG data for the left leg while section 2 contains the EMG data for the right alongside the force and trigger signal. The output file (rawemg) consists of 64 columns of EMG data i.e. 32 for the left leg and 32 from the right. The first 200 points are omitted as these contain an instrument driven series of very negative and irrelevant values.

```
for s=1:2%side
    for c=1:32%channel
        yy=(Signals(s).data{c,1}(1,200:end));
        rawemg(1:length(yy),c+(s-1)*32)=yy'; %#ok<SAGROW
    end
```

In the fourth and final section the force and trigger signal are extracted from channels 33 (left force), 34 (right force) and 35 (trigger) in the second subsection of the 'Signals' structure. The force data is processed to integrate it back to kg wt values using a coefficient calculated in a separate experiment.

```
if s==2
    Lff=(Signals(s).data{33,1}(1,200:end));
    LForce=(cumsum(Lff)/2048)*-614.7; % gives force in Kg wt nb need to check
    calc against Amelias data
    LForceList(f)=max(LForce);
    Rff=(Signals(s).data{34,1}(1,200:end));
    RForce=(cumsum(Rff)/2048)*-614.7; % gives force in Kg wt
    RForceList(f)=max(RForce);
    Trigger=(Signals(s).data{35,1}(1,200:end));
end
end
```

All outputs are appended to the original matfile

```
save([Root File], 'rawemg', 'LForce', 'RForce', 'Trigger', '-append')
end
```

9.2.2.2.3 Filter_Check_Nordic_CD

This program filters the EMG data for all channels and plots the filtered data overlaying the unfiltered data for comparison

The force and trigger data are also plotted

```
clear
close all hidden
Root='F:\PHD DATA\Study 2 - Nordic Activation\DATA\EXTRACTED EMG - Copy\REAL\';
DS=dir([Root '*.mat']);
Files={DS.name};

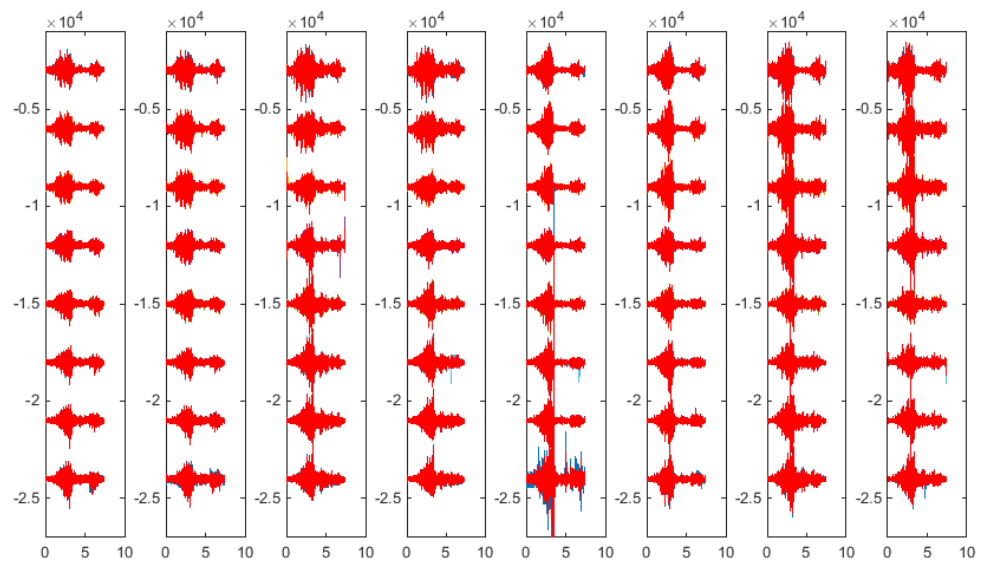
for f=1:length(Files)

    File=Files{f};
    load([Root File])
    figure; set(gcf, 'windowstyle', 'docked')
    clear FiltDat
    for p=1:8

        subplot(1,8,p)
```

The filter is set for a notch about 50Hz and a band pass @ 10-500hz

```
        for c=1:8
            yy=rawemg(:,c+(p-1)*8);
            Fq=2048;
            Time=((1:length(yy))-1)/Fq;
            Raw=timeseries(yy,Time);
            Ints=[48 52]; % the frequency intervals, in hertz, for filtering the
data:
            Band=[10 500];
            Filt = idealfilter(Raw,Ints,'notch');
            Filt1 = idealfilter(Filt,Band,'Pass');
            yyF=Filt1.Data;
            FiltDat(:,c+(p-1)*8)=yyF; %#ok<SAGROW>
            plot(Time, yy-3000*c);
            hold on
            plot(Time, yyF-3000*c, 'r');
            ylim([-27000,-1000]);
            text(length(yyF),yyF(end)+c*-3000,int2str(c))
        end
    end
```

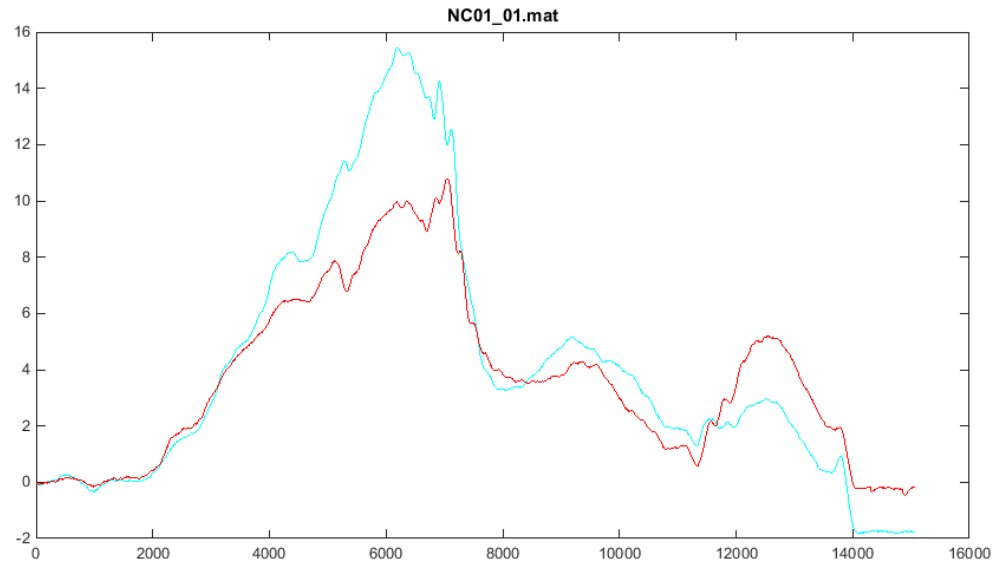
```
end
```

```
title([File], 'interpreter', 'none', 'VerticalAlignment', 'Bottom')
```

```
save([Root File], 'FiltDat', '-append')
```

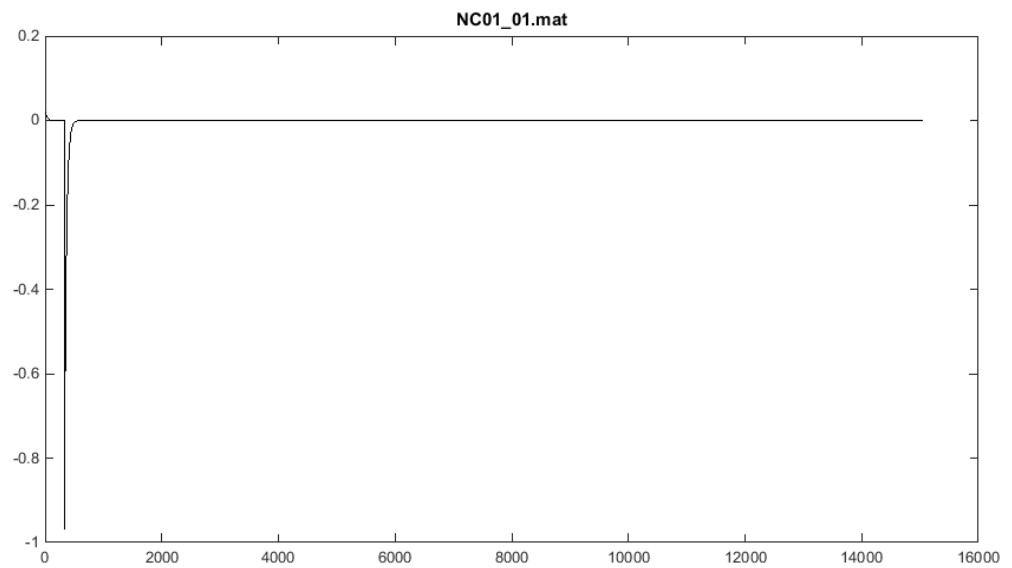
Graph the force

```
figure; set(gcf, 'windowstyle', 'docked')
plot(LForce, 'c')
hold on
plot(RForce, 'r')
title([File], 'interpreter', 'none', 'VerticalAlignment', 'Bottom')
```



Graph the trigger

```
figure; set(gcf, 'windowstyle', 'docked')
plot(Trigger, 'k')
title([File], 'interpreter', 'none', 'VerticalAlignment', 'Bottom')
```



end

9.2.2.2.4 GrEq_Nordic_CD

This program calculate the regional contribution of channels to overall maximal activity in that limb

The file location is identified. There is an option to plot the results over the entire contraction

```
clear
Plot=1; % set to 1 to make movies
Root='F:\PHD DATA\Study 2 - Nordic Activation\DATA\EXTRACTED EMG - Copy\Real\';
DS=dir([Root '*.mat']);
Files={DS.name};
```

This loop extracts and arranges the EMG channels according to side per subject

```
for f=1:length(Files)
    File=Files{f};
    load([Root File])
    figure(1); set(gcf, 'windowstyle', 'docked'); clf
    Time=(1:length(FiltDat))/2048;
    LeftChans=1:32;
    RightChans=33:64;
```

This is the calculation for the left side: The average activity across all left channels is calculated and smoothed of a 500ms window

```
MeanActL=nanmean(abs(FiltDat(:, LeftChans)),2);
% The averaged signal is smoothed across 500ms window to eliminate momentary peaks
MeanActLS=smooth(MeanActL,500);
% The timepoints where the average smoothed signal is within 20% of the max
% of this signal is identified
Luse=MeanActLS>0.8*nanmax(MeanActLS);
% The mean activity during this time for each channel is calculated
LChMax=nanmean(abs(FiltDat(Luse, :)));
% The total activity of all channels during this time is calculated
LSum=nansum(LChMax(LeftChans));
% The total activity is divided by the mean channel activity to give the relative
channel activity.
LChMaxRel=LChMax/LSum;
LBiggest=nanmax(LChMaxRel);
```

This process is repeated for the right side

```
MeanActR=nanmean(abs(FiltDat(:, RightChans)),2);
```

```
% average across all right channels
MeanActRS=smooth(MeanActR,500);
Ruse=MeanActRS>0.8*nanmax(MeanActRS);
RChMax=nanmean(abs(FiltDat(Ruse, :)));
RSum=nansum(LChMax(RightChans));
RChMaxRel=RChMax/RSum;
RBiggest=nanmax(RChMaxRel);
```

This is the optional plotting function. Channels are arranged according to their anatomical location on the leg. The activity of each channel is plotted over 101 time points and the relative activity as a red bar.

```
q=0;
if plot
for ns=1:102:length(Time)-102
    q=q+1;
    bit=FiltDat(ns:ns+101,:);
    FD=abs(bit);
    mLFD=mean(FD)/sum(LChMax);
    mRFD=mean(FD)/sum(RChMax);
    for p=1:8
        s=[5 6 7 8 4 3 2 1]; % order of ch set for picture to equal anatomy
        subplot(1,8,p)
        cla;
        if (LeftChans && p<5) || (~LeftChans && p>4);
            yyL=LChMaxRel((1:8)+(s(p)-1)*8);
            yyR=RChMaxRel((1:8)+(s(p)-1)*8);
            hb=barh(8:-1:1,yy);
            set(hb, 'EdgeColor', 'r', 'FaceColor', 'w', 'Barwidth', 1)
            hold on
        end
        yyL=mLFD((1:8)+(s(p)-1)*8);
        yyR=mRFD((1:8)+(s(p)-1)*8);
        barh(8:-1:1,yy)
        xlim([0 LBiggest+0.005])
        set(gca, 'YTickLabel', {'8' '7' '6' '5' '4' '3' '2' '1'})
    end
end
drawnow
end
end
```

The data is converted to column array for export

```
LCMaxRelXLS=LChMaxRel(1,1:32)';
RCMaxRelXLS=RChMaxRel(1,33:64)';
```

```

    save ([Root File], 'LChMax', 'RChMax', 'LSum', 'RSum', 'LChMaxRel', 'RChMaxRel',
        'LCMaxRelXLS', 'RCMaxRelXLS', '-append')

```

```

end

```

9.2.2.2.5 NordicExcelExport_CD

This program exports the relative activation data to an excel file.

```

clear
%parpool
Root='F:\PHD DATA\Study 2 - Nordic Activation\DATA\EXTRACTED EMG -
Copy\Reliability\';
DS=dir([Root '*.mat']);
Files={DS.name};
exceloutput = 'NordicRELIABILITYChMaxReloutput.xlsx';
warning('off', 'MATLAB:xlswrite:AddSheet')

for f=1:length(Files)
    File=Files{f};
    load([Root File])
    xlswrite(exceloutput,LCMaxRelXLS,File,'B3')
    xlswrite(exceloutput,RCMaxRelXLS,File,'C3')
end

```

9.2.2.2.6 NordicZero2Nan_CD

This program converts any zeros to Nans in the EMG record and resaves the file

```

clear
Root='F:\PHD DATA\Study 2 - Nordic Activation\DATA\EXTRACTED EMG - Copy\Real\';
DS=dir([Root '*.mat']);
Files={DS.name};

for f=1:length(Files)
    File=Files{f};
    load([Root File])
    FiltDat(FiltDat==0)=nan;
end

save ([Root File], 'FiltDat', '-append');

```

9.2.2.2.7 Fq_ForceOn_Off_CD

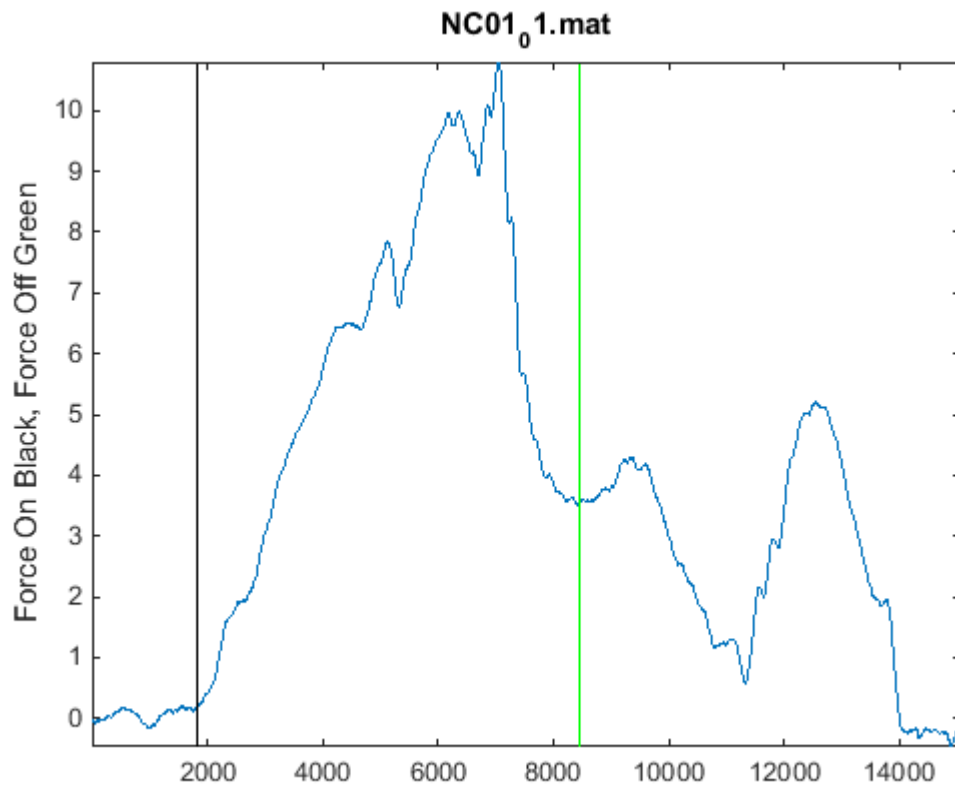
This program is used to extract the portion of the record where the strain gauges at the ankles are under load. This is performed via visual inspection using the MarkFqForce function

```
clear

Root='F:\PHD DATA\Study 2 - Nordic Activation\DATA\EXTRACTED EMG - Copy\REAL\';
DS=dir([Root '*.mat']);
Files={DS.name};

for f=1:length(Files);
    File=Files{f};
    load([Root File])
    LFqForceOn=[]; LFqForceOff=[]; RFqForceOn=[]; RFqForceOff=[];
    disp([ File ' LeftForce']);
    Title=[File ' LeftForce'];
    [LFqForceOn, LFqForceOff]=MarkFqForce(LForce, LFqForceOn, LFqForceOff, File);
    save ([Root File], 'LFqForceOn', 'LFqForceOff', '-append')

    disp([ File ' RightForce']);
    Title=[File ' RightForce'];
    [RFqForceOn, RFqForceOff]=MarkFqForce(RForce, RFqForceOn, RFqForceOff, File);
    save ([Root File], 'RFqForceOn', 'RFqForceOff', '-append')
end
```



9.2.2.2.8 RMSEMG_Nordic_CD

This program performs an RMS calculation using a 102hz (i.e. 50ms) non overlapping window using the rms function. Data for RMS conversion is extracted during the time of loading using the force on/force off timepoints. The original data is in microvolts so is divided by 1000 to convert to millivolts.

```
clear

Root='F:\PHD DATA\Study 2 - Nordic Activation\DATA\EXTRACTED EMG - Copy\reAL\';
DS=dir([Root '*.mat']);
Files={DS.name};

for f=1:length(Files);

    File=Files{f};
    load([Root File])
    LFiltDat=abs(FiltDat(:,1:32));
    RFiltDat=abs(FiltDat(:,33:64));
    LRoundOn=round(LFqForceOn);
    LRoundOff=round(LFqForceOff);
    RRoundOn=round(RFqForceOn);
    RRoundOff=round(RFqForceOff);
```

```
LRMSEMG=[]; LEMG=[]; useLRMSEMG=[]; LEMGMV=[];
RRMSEMG=[]; REMG=[]; useRRMSEMG=[]; REMGMV=[];
```

Calculations are performed for the left and right side separately

```
for p=1:32
    LRMSEMG=LFilterDat((LRoundOn:LRoundOff),:);
    useLRMSEMG=rms((LRMSEMG(:,p)),102,0,1);
    useLRMSEMG=useLRMSEMG';
    for i=1:length(useLRMSEMG);
        LEMG(:,p)=[useLRMSEMG(i,:)];
    end
    LEMGMV=LEMG/1000;
end

for p=1:32
    RRMSEMG=RFilterDat((RRoundOn:RRoundOff),:);
    useRRMSEMG=rms((RRMSEMG(:,p)),102,0,1);
    useRRMSEMG=useRRMSEMG';
    for i=1:length(useRRMSEMG);
        REMG(:,p)=[useRRMSEMG(i,:)];
    end
    REMGMV=REMG/1000;
end

save ([Root File], 'LEMGV', 'REMGV', '-append')

end
```

9.2.2.2.9 Rms

This function was obtained on the matlab community forum site.

Calculates windowed (over- and non-overlapping) RMS of a signal using the specified windowlength $y = \text{rms}(\text{signal}, \text{windowlength}, \text{overlap}, \text{zeropad})$ signal is a 1-D vector windowlength is an integer length of the RMS window in samples overlap is the number of samples to overlap adjacent windows (enter 0 to use non-overlapping windows) zeropad is a flag for zero padding the end of your data...(0 for NO, 1 for YES) ex. $y = \text{rms}(\text{mysignal}, 30, 10, 1)$. Calculate RMS with window of length 30 samples, overlapped by 10 samples each, and zeropad the last window if necessary ex. $y = \text{rms}(\text{mysignal}, 30, 0, 0)$. Calculate RMS with window of length 30 samples, no overlapping samples, and do not zeropad the last window

Author: A. Bolu Ajiboye

```
function y = rms(signal, windowlength, overlap, zeropad)
```



```
delta = windowlength - overlap;
```

Error using rms (line 17)
Not enough input arguments.

Calculate RMS

```
indices = 1:delta:length(signal);  
% Zeropad signal  
if length(signal) - indices(end) + 1 < windowlength  
    if zeropad  
        signal(end+1:indices(end)+windowlength-1) = 0;  
    else  
        indices = indices(1:find(indices+windowlength-1 <= length(signal), 1,  
'last'));  
    end  
end  
  
y = zeros(1, length(indices));  
% Square the samples  
signal = signal.^2;  
  
index = 0;  
for i = indices  
    index = index+1;  
    % Average and take the square root of each window  
    y(index) = sqrt(mean(signal(i:i+windowlength-1)));  
end
```

9.2.2.2.10 Nordic_NormRMSEMGCalc_CD

This program calculates the normalised RMS signal. The mean signal for each channel is calculated and the percentage of the RMS amplitude calculated in relation to this.

```
clear  
  
Root='F:\PHD DATA\Study 2 - Nordic Activation\DATA\EXTRACTED EMG - Copy\REAL\';  
DS=dir([Root '*.mat']);  
Files={DS.name};  
  
for f=1:length(Files);  
    File=Files{f};  
    load([Root File])  
    MeanLRMSEMG=[]; NormLRMSEMG=[]; LNormRMSEMG=[];  
    MeanRRMSEMG=[]; NormLRMSEMG=[]; RNormRMSEMG=[];
```

This calculation is performed for the left channels

```
for p=1:32
    MeanLRMSEMG=mean(LEMGMV(:,p));
    NormLRMSEMG=(LEMGMV(:,p)/MeanLRMSEMG)*100;
    for i=1:length(NormLRMSEMG);
        LNormRMSEMG(:,p)=[NormLRMSEMG(:,1)];
    end
end
```

This calculation is performed for the right channels

```
for p=1:32
    MeanRRMSEMG=mean(REMGMV(:,p));
    NormRRMSEMG=(REMGMV(:,p)/MeanRRMSEMG)*100;
    for i=1:length(NormRRMSEMG);
        RNormRMSEMG(:,p)=[NormRRMSEMG(:,1)];
    end
end
```

And the data is saved to the matfile

```
save ([Root File], 'LNormRMSEMG', 'RNormRMSEMG', '-append')
end
```

9.2.2.2.11 NordicPeakNormEMG_CD

This program calculates the peak of the normalised RMS EMG signal

```
clear
Root='F:\PHD DATA\Study 2 - Nordic Activation\DATA\EXTRACTED EMG - Copy\Real\';
DS=dir([Root '*.mat']);
Files={DS.name};

for f=1:length(Files)
    File=Files{f};
    load([Root File])
    PeakNormL=max(LNormRMSEMG(:,:));
    PeakNormR=max(RNormRMSEMG(:,:));
    save ([Root File], 'PeakNormL', 'PeakNormR', '-append')
end
```

9.2.2.2.12 NordicPeakNormExcel_CD

This program exports the peak of the normalised RMS EMG signal for each channel to excel.

```
clear
Root='F:\PHD DATA\Study 2 - Nordic Activation\DATA\EXTRACTED EMG - Copy\Real\';
DS=dir([Root '*.mat']);
Files={DS.name};
exceloutput = 'NordicRealPeakNormEMGOutput.xlsx';
warning('off','MATLAB:xlswrite:AddSheet')

for f=1%34:length(Files); %33 is missing force
    File=Files{f};
    load([Root File])
    LPeakNormXLS=PeakNormL';
    RPeakNormXLS=PeakNormR';
    xlswrite(exceloutput,LPeakNormXLS,File,'B3')
    xlswrite(exceloutput,RPeakNormXLS,File,'C3')
end
```

9.2.2.2.13 Interpolate_for_iEMG_Nordic_CD

This program performs a resampling of the normalised RMS signals for each channel to time standardised the length of the records according to the longest duration of loading across all subjects. The durations were examined separately

```
clear

Root='F:\PHD DATA\Study 2 - Nordic Activation\DATA\EXTRACTED EMG - Copy\reliability\';
DS=dir([Root '*.mat']);
Files={DS.name};

for f=1:length(Files);
    File=Files{f};
    load([Root File])
    LRMSEMGInt=[];
    RRMSEMGInt=[];

    for j=length(LNormRMSEMG);
        p=1:32;
        LRMSEMGInt=resample(LNormRMSEMG(:,p),243,j); %for real data
        %LRMSEMGInt=resample(LNormRMSEMG(:,p),173,j); %for reliability data
    end

    for j=length(RNormRMSEMG);
```

```

        p=1:32;
        RRMSEMGInt=resample(RNormRMSEMG(:,p),248,j);%for real data
        %RRMSEMGInt=resample(RNormRMSEMG(:,p),152,j);
    end

    save ([Root File], 'LRMSEMGInt', 'RRMSEMGInt', '-append')
end

```

9.2.2.2.14 iEMG_Nordic_CD

This program calculated the area under the curve of the time normalised RMS signals for each channel and subject using the 'trapz' command.

```

clear

Root='F:\PHD DATA\Study 2 - Nordic Activation\DATA\EXTRACTED EMG - Copy\real\';
DS=dir([Root '*.mat']);
Files={DS.name};

for f=1:length(Files);
    File=Files{f};
    load([Root File])
    LiEMGCalc=[]; LiEMG=[];
    RiEMGCalc=[]; RiEMG=[];
    for p=1:32
        LiEMGCalc=(trapz(LRMSEMGInt(:,p)))/1000;
        for i=1:length(LiEMGCalc);
            LiEMG(:,p)=LiEMGCalc(:,:);
        end
    end
    for p=1:32
        RiEMGCalc=(trapz(RRMSEMGInt(:,p)))/1000;
        for i=1:length(RiEMGCalc);
            RiEMG(:,p)=RiEMGCalc(:,:);
        end
    end
    save ([Root File], 'LiEMG', 'RiEMG', '-append')
end

```

9.2.2.2.15 iEMG_Nordic_Excel_CD

This program exports the iEMG data for each channel to an excel document

```

clear

Root='F:\PHD DATA\Study 2 - Nordic Activation\DATA\EXTRACTED EMG - Copy\real\';
DS=dir([Root '*.mat']);
Files={DS.name};

```

```

exceloutput = 'NordicEMGRelOutput.xlsx';
warning('off','MATLAB:xlswrite:AddSheet')

for f=1:length(Files);
    File=Files{f};
    load([Root File])
    LiEMGExcel=LiEMG';
    RiEMGExcel=RiEMG';
    xlswrite(exceloutput,LiEMGExcel,File,'B3')
    xlswrite(exceloutput,RiEMGExcel,File,'C3')
end

```

9.2.2.2.16 Nordic_MDF_CD

This program is used to extract the median EMG frequency for each channel during the Nordic loading. The code for calculating the median frequency were downloaded from the Matlab fileshare site

```

clear

Root='F:\PHD DATA\Study 2 - Nordic Activation\DATA\EXTRACTED EMG - Copy\real\';
DS=dir([Root '*.mat']);
Files={DS.name};

for f=1:length(Files);

    File=Files{f};
    load([Root File])
    LMDF=[]; RMDF=[];

```

Calculate median frequency for the left side for all available channels

```

for p=1:32
    Ls = useLFiltDat(:,p);
    if Ls==0
        continue
    else
        Fs = 2048; % Sampling
        Frequency
        Fn = Fs/2; % Nyquist
        Frequency
        LL = length(useLFiltDat);

```

The fft (Fast Fourier Transform) function in the matlab signals toolbox is used to extract the frequency spectra of the signal

```

LFTs = fft(Ls)/LL;
LFv = linspace(0, 1, fix(LL/2)+1)*Fn;           % Frequency
Vector
LIV = 1:length(LFv);                             % Index Vector
LCumAmp = cumtrapz(LFv, abs(LFTs(LIV)));         % Integrate
FFT Amplitude
LMedFreq = interp1(LCumAmp, LFv, LCumAmp(end)/2); % Use '
interp1' To Find 'MF'

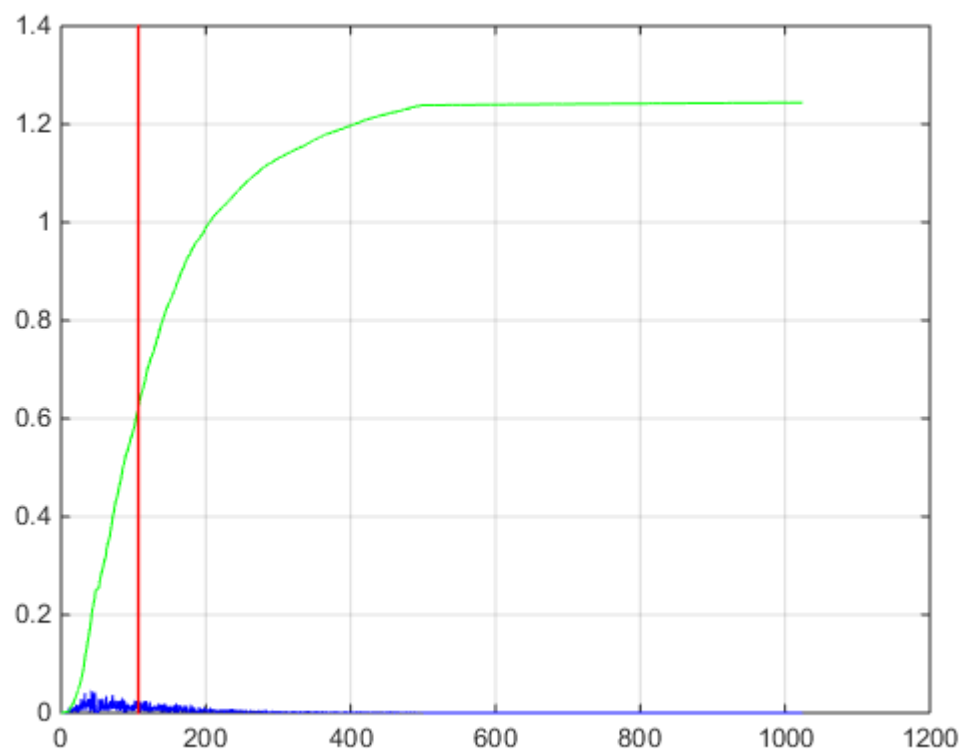
```

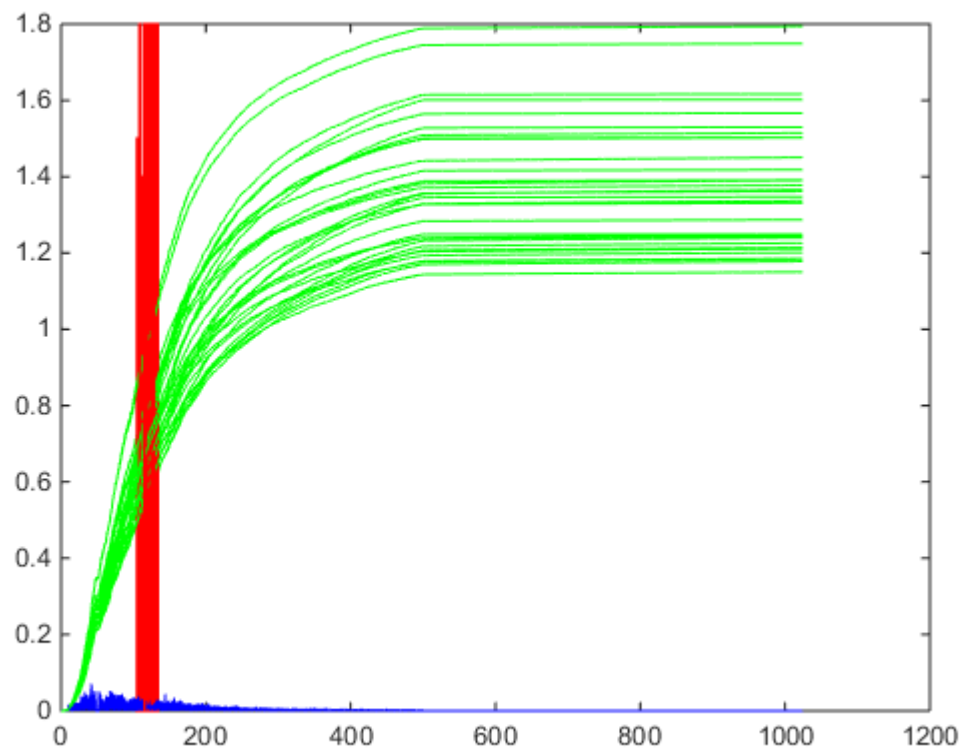
The results are plotted for all 32 channels

```

figure(f)
plot(LFv, abs(LFTs(LIV))*2, '-b')                % Plot FFT
hold on
plot(LFv, LCumAmp, '-g')                         % Plot
Cumulative Amplitude Integral
plot([LMedFreq LMedFreq], ylim, '-r', 'Linewidth',1) % Plot Median
Frequency
grid
LMDF(:,p)=[LMedFreq(:,:)];

```





```

end
end

```

The same process is completed for the right limb.

```

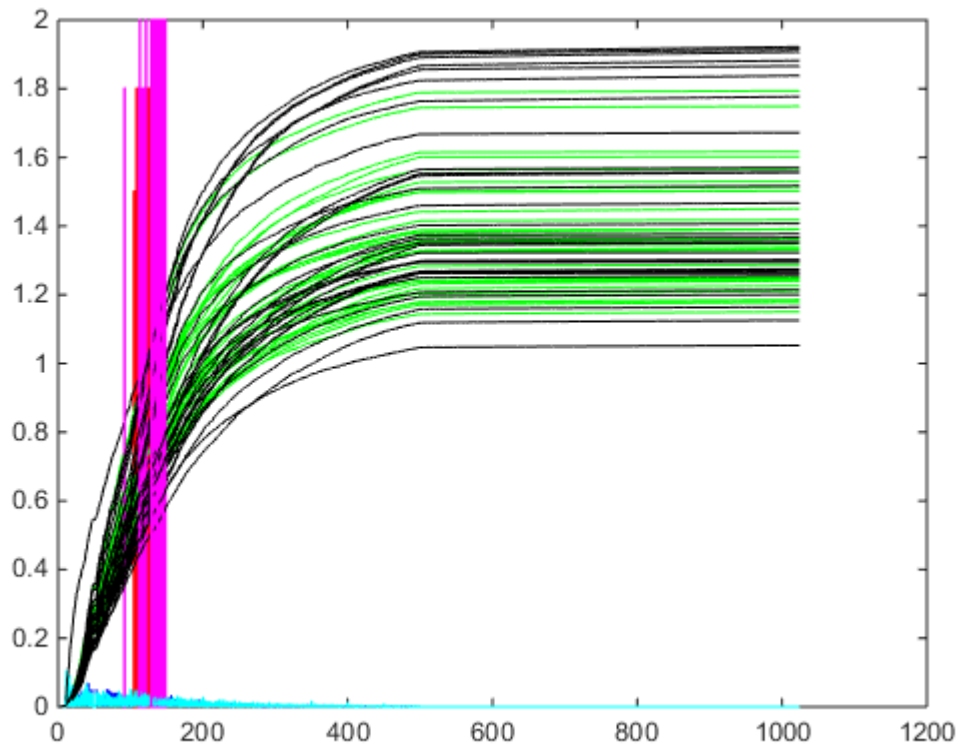
for p=1:32
    Rs = userFiltDat(:,p);
    if RS==0
        continue
    else
        Fs = 2048;                                % Sampling
        Frequency
        Fn = Fs/2;                                % Nyquist
        Frequency
        RL = length(userFiltDat);
        RFTs = fft(Rs)/RL;
        RFv = linspace(0, 1, fix(RL/2)+1)*Fn;      % Frequency
        Vector
        RIV = 1:length(RFv);                       % Index Vector
        RCumAmp = cumtrapz(RFv, abs(RFTs(RIV)));    % Integrate
        FFT Amplitude
        RMedFreq = interp1(RCumAmp, RFv, RCumAmp(end)/2); % Use '
        interp1' To Find 'MF'
    end
end

```

```

plot(RFv, abs(RFTs(Rlv))*2, '-c')           % Plot FFT
hold on
plot(RFv, RCumAmp, '-k')                   % Plot Cumulative Amplitude Integral
plot([RMedFreq RMedFreq], ylim, '-m', 'LineWidth',1) % Plot Median Frequency
RMDF(:,p)=[RMedFreq(:,:)];
end
end

```



The median frequency for channels of the right and left limb are saved to the matfile.

```

save ([Root File], 'LMDF', 'RMDF', '-append')
end

```

9.2.2.2.17 MDF_Nordic_Excel_CD

This program extracts and exports the MDF data for each channel to an excel document. The left and right sides are stored on a separate sheet for each subject


```
clear
Root='F:\PHD DATA\Study 2 - Nordic Activation\DATA\EXTRACTED EMG -
Copy\Reliability\';
DS=dir([Root '*.mat']);
Files={DS.name};
exceloutput = 'NordicRelMDFOutput.xlsx';
warning('off','MATLAB:xlswrite:AddSheet')
```

The subject is selected and the MDF data transposed to column vector and saved to the corresponding sheet.

```
for f=1:length(Files);
    File=Files{f};
    load([Root File])
    LMDFExcel=LMDF';
    RMDFExcel=RMDF';
    xlswrite(exceloutput,LMDFExcel,File,'B3')
    xlswrite(exceloutput,RMDFExcel,File,'C3')
end
```

9.2.2.3 Force analysis

9.2.2.3.1 PeakForceExtract

This program calculates the peak force from the force data for each limb and exports to excel. Data for the left and right legs are stored on separate sheets for each subject trial.

```
clear
Root='F:\PHD DATA\Study 2 - Nordic Activation\DATA\EXTRACTED EMG -
Copy\RELIABILITY\';
DS=dir([Root '*.mat']);
Files={DS.name};
exceloutput = 'NordicRelPeakForceOutput.xlsx';
warning('off','MATLAB:xlswrite:AddSheet')
```

Peak force calculation performed

```
for f=1:length(Files);
    File=Files{f};
    load([Root File])
    PeakForceL=max(LForce);
    PeakForceR=max(RForce);
    save ([Root File], 'PeakForceL', 'PeakForceR', '-append')
end
```

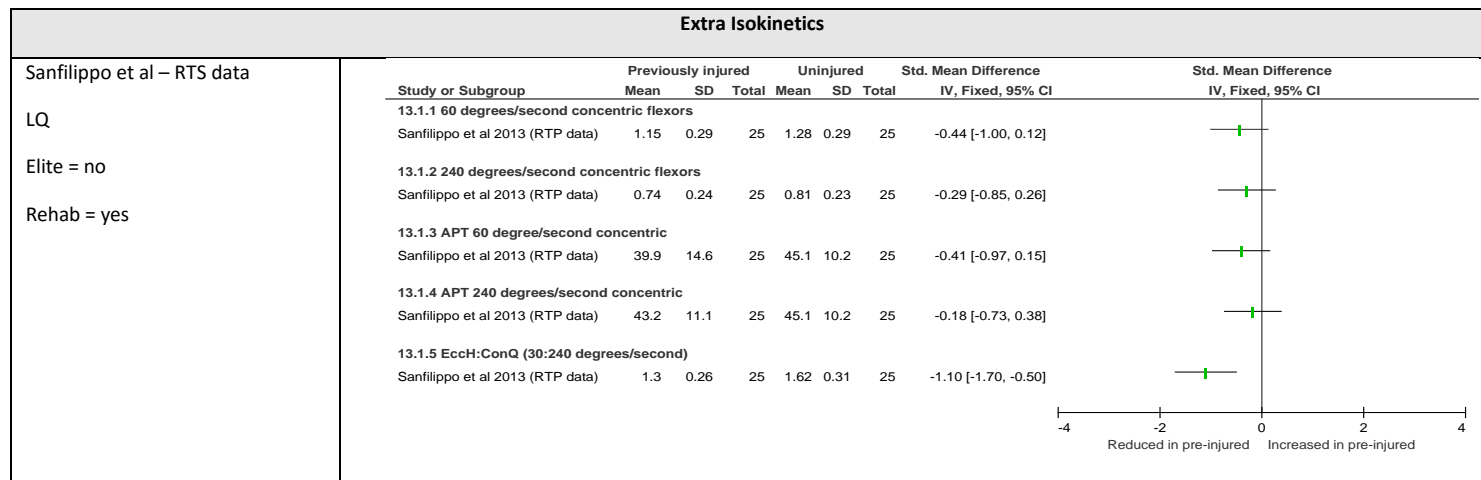
And exported

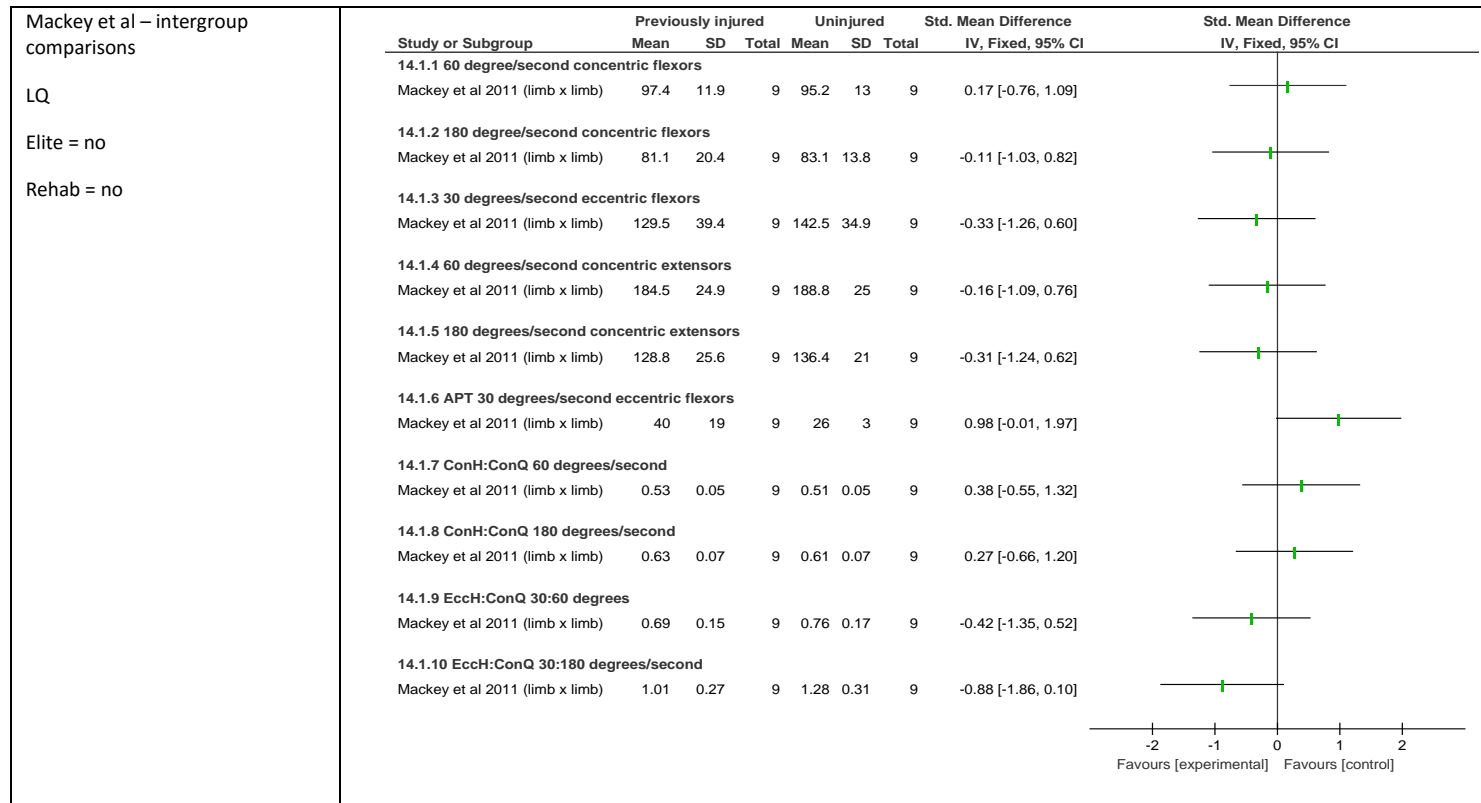
```
for f=1:length(Files);  
    File=Files{f};  
    load([Root File])  
    xlswrite(exceloutput,PeakForceL,File,'B3')  
    xlswrite(exceloutput,PeakForceR,File,'C3')  
end
```

9.3 Overview of findings – Systematic Review

ISOKINETICS						
OUTCOME	Standard Mean Difference INTERPRETATION	LEVEL OF EVIDENCE	SIGNIFICANCE (p)	HQ/MQ only	ELITE only	Complete REHAB only
Peak torque (concentric flexors)						
- 30°/second	One study: Medium (-1.12)	Very limited	One study	NA	NC (-1.12)	NA
- 60°/second	NS	Moderate	NS (0.36)	Single – NS	NS (2 studies)	NS (2 studies)
- 180°/second	NS	Moderate	NS (0.18)	NA	Single- NC	Single – NC
- 240°/second	One study: NS	Very limited	One study	NA	NA	Single – NC
- 270°/second	One study: NS	Very limited	One study	NA	NC	NA
- 300°/second	NS	Moderate	NS (0.97)	NA	NA	NA
Peak torque (eccentric flexors)						
- 30°/second	Medium (-0.62)	Moderate	P=0.03	NA	Single (-1.59)	NA
- 60°/second	NS	Moderate	NS (0.18)	NA	Single - NC	Single – NC
- 180°/second	NS	Limited	NS (0.62)	NA	NS (v limited)	Single – NC
- 230°/second	One study: Large (-1.84)	Very limited	One study	NA	NS	NA
- 300°/second	One study: Medium (-0.75)	Very limited	One study	NA	NA	NA
Peak torque (concentric exten)						
- 30°/second	One study: Medium (-1.16)	Very limited	One study	NA	Single- NC	NA
- 60°/second	NS	Moderate	NS (0.58)	NA	NC (2 studies)	NA
- 180°/second	NS	Moderate	NS (0.76)	NA	Single- NC	NA
- 270°/second	One study: Small (-0.48)	Very limited	One study	NA	Single- NC	NA
- 300°/second	NS	Moderate	NS (0.08)	NA	NA	NA

Angle of peak torque (concentric)						
- 60°/second	NS	Limited	NS (=0.15)	NA	Single (1.54)	Single – NC
- 240°/second	One study (NS)	Very limited	One study	NA	NA	Single - NC
Angle of peak torque (eccentric)						
- 30°/second	One study (Medium 0.77)	Very limited	One study	NA	NA	Single – NC
Conventional ratio (H:Q)						
- 30°/second	One study (NS)	Very limited	One study	NA	Single- NC	NA
- 60°/second	Small (-0.34)	Moderate	P=0.002	NS (P=0.23)	NS (-0.21)	NA
- 120°/second	One study (NS)	Very limited	One study	NA	Single - NC	NA
- 180°/second	NS	Moderate	NS (0.51)	NA	Single – NC	NA
- 300°/second	NS	Moderate	NS (0.5)	NA	NA	NA
Functional ratio (H:Q)						
- 30°/second: 60°/second	One study (NS)	Very limited	One study	NA	NA	NA
- 30°/second: 180°/second	One study (NS)	Very limited	One study	NA	NA	NA
- 30°/second: 240°/second	Medium (-0.62)	Moderate	P=0.0002	NA	-0.78 P=0.0002	Single – NS
- 60°/second: 60°/second	NS	Moderate	NS (0.06)	Single = NS	-0.5 P=0.04	NA
- 180°/second: 180°/second	One study: NS	Very limited	One study	Single = NS	Single – NC	NA





OTHER FORCE RELATED MEASURES						
OUTCOME	SMD INTERPRETATION	LEVEL OF EVIDENCE	SIGNIFICANCE	SENSITIVITY (HQ/MQ)	ELITE	REHAB
KINETICS						
Treadmill running						
- Horizontal force @ centre of mass (within group)	Large reduction (-3.81)	Very limited	Single paper	NA	NC	NA
- Horizontal force @ centre of mass (v controls)	Large reduction (>-2.23)	Very limited	Single paper	NA	NC	NA
- Horizontal/vertical ground reaction force (within group)	Non-significant	Very limited	Single paper	NA	NC	NA
Inverse dynamics						
- Peak moments/powers @ hip and knee	Non-significant	Very limited	Single paper	NA	NA	NA
Radar based outcomes						
- Peak power	Medium reduction (-0.93)	Very Limited	Single paper	NC	NC	NA
- Peak horizontal force	Medium reduction (--0.81)	Very Limited	Single paper	NC	NC	NA
NORDBORD						
Inter-limb (injured group only)						
- Max force	Small reduction (-0.38)	Moderate	P=0.05 (borderline)	NS P=0.17	NC	NS P=0.16
Intra-group (v controls)						
- Max force	Medium (-0.64)	Mod. (v right)	P=0.0002	NS P=0.08	NC	NS (P=0.08)
	Small reduction (-0.45)	Mod (v left)	P=0.009	NS P=0.57	NC	NS (P=0.57)
Change in pre-season strength:						
- Injured v controls gains	Small reduction (-0.54)	V limited	P=0.05 (borderline)	NC	NC	NC

MOVEMENT BEASED MEASURES						
OUTCOME	SMD INTERPRETATION	LEVEL OF EVIDENCE	SIGNIFICANCE	SENSITIVITY (HQ/MQ)	ELITE	REHAB
TEMPORAL						
Radar based outcomes						
- Time to reach 5m (Men)	Medium increase (1.06)	Very limited	Single paper	NC	NC	NA
- Contact times (Brughelli)						
versus contra	NS	Very limited	Single paper	NA	NC	NA
versus left control	NS (0.1)	Very limited	Single paper	NA	NC	NA
versus right control	NS (-0.57)	Very limited	Single paper	NA	NC	NA
- Movement reaction (Sole 2012)	SMD not calculable – faster on previously injured side: -54ms (Range -108 to 0),	Very limited	Single paper	NA	NA	NA
KINEMATIC						
3D kinematics						
- Movement symmetry (Daly)						
Pelvis (late swing)	SMD not calculable – increase in anterior tilt	Very Limited	Single paper	NA	NC	NA
Hip (late swing)	SMD not calculable – increase in flexion	Very Limited	Single paper	NA	NC	NA
Knee (contact)	SMD not calculable – increase in medial rotation	Very Limited	Single paper	NA	NC	NA
- Movement ampl (Lee)						
Hip fxn (late swing)	Small reduction (-0.35)	Very limited	Single paper	NA	NA	NA
Kn ext (late swing)	Non-significant	Very limited	Single paper	NA	NA	NA
- COM displacement (Brughelli)	SMD not calculable – no change	Very limited	Single paper	NA	NC	NA
- MT length (Silder 2010)	No change	Very limited	Single paper	NA	NA	NA
- Movement reactions (Sole 2012)	SMD not calculable – faster initiation of movement	Very limited	Single paper	NA	NA	NA

ELECTROMYOGRAPHIC						
OUTCOME	SMD INTERPRETATION	LEVEL OF EVIDENCE	SIGNIFICANCE	SENSITIVITY (HQ/MQ)	ELITE	REHAB
EMG ratio data (Daly 2015) (BF v) - Ipsilateral Gluteus Maximus - Ipsilateral Erector Spinae - Ipsilateral External Oblique - Contralateral Rectus Femoris	SMD not calculable – reduced SMD not calculable – reduced SMD not calculable – reduced SMD not calculable – reduced	Very limited Very limited Very limited Very limited	Single paper Single paper Single paper Single paper	NA NA NA NA	NC NC NC NC	NA NA NA NA
EMG amplitude @ IC, Propulsion, ES, TS) normalized to mean activity over stride (Silder 2010) : - BF, MH, VL -	SMD not calculable – no change SMD not calculable – no change	Very limited Very limited	Single paper Single paper	NA NA	NA NA	NA NA
EMG onset timings @ IC, Propulsion, ES, TS) (Silder 2010) - BF, MH, VL	Large reduction (@ 60° SMD = -1.54) (@ 180° SMD = -1.45)	Very limited	Single paper	NA	NA	NC
EMG max (normalized to MVC) 60 and 180°/second concentric and eccentric IKED (Opar 2013)	NS	Very limited	Single paper	NA	NA	NC
EMG median frequency at 60 and 180°/second concentric and eccentric contractions (Opar 2013)	NS	Very limited	Single paper	NA	NA	NC
iEMG @ 100ms post onset during 60°/second eccentric contraction (Opar 2013) - BF	Small increase in asymmetry (0.38)	Very Limited	Single paper	NA	NA	NC
iEMG @ 100ms post onset during 180°/second eccentric contraction (Opar 2013) - BF	Reduced (outer range) Reduced (outer range)	Very limited Very limited	Single paper Single paper	NA NA	NA NA	NA NA
RMS EMG normalised to peak during eccentric isokinetics @ 60°/second subdivided across quartiles (Sole 2011): - BF - MH	SMD not calculable – reduced SMD not calculable – reduced	Very Limited Very Limited	Single paper Single paper	NA NA	NA NA	NA NA
Onset times during double to single leg standing (Sole 2012) - Injured limb v average controls o BF o MH - Uninjured limb v average controls o BF o MH	SMD not calculable – reduced SMD not calculable – reduced SMD not calculable – reduced SMD not calculable – reduced	Very Limited Very Limited Very Limited Very Limited	Single paper Single paper Single paper Single paper	NA NA NA NA	NA NA NA NA	NA NA NA NA

9.4 Ethics approvals

9.4.1 Observational study 1



UCD Office of Research Ethics

UCD Sciences South
University College Dublin
Belfield, Dublin 4, Ireland

T +353 1 718 2387

An Oifig Éire Thairghe UCD

Ionad Eolaíochta UCD (Theas)
An Coláiste Ollscoile, Baile Átha Cliath
Belfield, Baile Átha Cliath 4, Éire

hrec@ucd.ie
www.ucd.ie/researchethics

April 10th, 2013

Mr Colm Daly
c/o Dr Ulrick McCarthy-Persson
UCD School of Public Health, Physiotherapy & Population Science,
Health Sciences Centre
Belfield
Dublin 4

Re: *LS-13-10-Daly-McCarthy-Persson: The Biomechanics of Running in Athletes with Previous Hamstring Injury*

Dear Mr Daly

Thank you for your recent submission for ethical review to the Human Research Ethics Committee – Sciences. Your application was reviewed at the meeting held on April 10th. The Decision of the Committee is to grant approval for this application which is subject to the conditions set out below.

Please note that approval is for the work and the time period specified in the above protocol and is subject to the following:

- If applicable, all permissions to access participants, whether internal (heads of Schools/Registrar) or external are obtained before the recruitment of the participants is commenced;
- Any amendments or requests to extend the original approved study will need to be approved by the Committee. Therefore you will need to submit by email the *Request to Amend/Extend Form* (HREC Doc 10);
- Any unexpected adverse events that occur during the conduct of your research should be notified to the Committee. Therefore you will need to Submit, by email, an *Unexpected Adverse Events Report* (HREC Doc 11);
- You or your supervisor (if applicable) are required to submit a signed *End of Study Report Form* (HREC Doc 12) to the Committee upon the completion of your study;

—/—

- This approval is granted on condition that you ensure that, in compliance with the Data Protection Acts 1988 and 2003. If applicable, all data will be destroyed in accordance with your application and that you will confirm this in your *End of Study Report* (HREC Doc 12), or indicate when this will occur and how this will be communicated to the Human Research Ethics Committee;
- You may require copies of submitted documentation relating to this approved application and therefore we advise that you retain copies for your own records;
- Please note that the granting of this ethical approval is premised on the assumption that the research will be carried out within the limits of the law;
- Please also note that approved applications and any subsequent amendments are subject to a Research Ethics Compliance Review.

The Committee wishes you well with your research and look forward to receiving your End of Study Report. All forms are available on the website www.ucd.ie/researchethics please ensure that you submit the latest version of the relevant form. If you have any queries regarding the above please contact the Office of Research Ethics and please quote your reference in all correspondence.

Yours sincerely,



Professor William Watson
Chair, Human Research Ethics Committee - Sciences

c/o Dr Dylan Morrissey
Department of Sports Medicine
Mile End Hospital
Bancroft Road
London E1 4NS

21st May 2013

To Whom It May Concern: Ethical Approval Confirmation

Re: The Biomechanics of Running in Athletes with Previous Hamstring Injury

The above study was viewed under the aegis of the generic lab based approval of: **QMREC2011/07– Human Performance Measurement – A Generic Ethics Application**

This individual study was assessed, approved, and registered on the 23rd and 24th of January 2013.

This approval is valid for a period of two years, (if the study is not started before this date then the applicant will have to reapply to the Committee).

Yours faithfully



Ms Elizabeth Hall – QMREC Chair.

9.4.2 Observational study 2

FW: Amended Ethics Application

Dylan Morrissey

Mon 03/02/2014 11:23

To: Hazel Covill <h.covill@qmul.ac.uk>; Colm Daly <c.daly@qmul.ac.uk>;

📎 1 attachments (130 KB)
Nordic Ethics Application.doc

Dear Colm,
Thank you for this – I can now confirm it is approved. I note you will not recruit until you have agreement letters for any given club, that have been seen by me and stored.

Hazel, please note this is approved under the HPL ethics.

Best wishes
Dylan

Thanks for follow @DrDylanM

Dr Dylan Morrissey
Senior Clinical Lecturer and Consultant Physiotherapist
Centre for Sports and Exercise Medicine
William Harvey Research Institute
Bart's and the London School of Medicine and Dentistry
Queen Mary University of London

a: Mile End Hospital, Bancroft road, London E1 4DG
t: +447941710273
e: d.morrissey@qmul.ac.uk

9.4.3 Case reports

another hammi study

Dylan Morrissey

Wed 17/09/2014 11:44

To: Colm Daly <c.daly@qmul.ac.uk>; Hazel Covill <h.covill@qmul.ac.uk>;

1 attachments (129 KB)

Serial Measures Ethics DM.doc

Colm,

I am happy to approve this study under the generic lab ethics as study number QMREC2014/24/20.

Hazel – fyi. Please note this may well involve professional footballers, notable for insurance purposes.

Best wishes

Dylan

Thanks for follow @DrDylanM

Dr Dylan Morrissey

NIHR/HEE Senior Clinical Lecturer and Consultant Physiotherapist

Centre for Sports and Exercise Medicine

William Harvey Research Institute

Bart's and the London School of Medicine and Dentistry

Queen Mary University of London


a: Mile End Hospital, Bancroft road, London E1 4DG

t: +447941710273

e: d.morrissey@qmul.ac.uk

9.5 Ethics Applications/Documentation

9.5.1 Observational study 1 (Running)

<p>Official use only – HREC Reference: V: July 2011</p>	<p>Date Received:</p>
<div style="display: flex; align-items: center; justify-content: center;"><div>Human Subjects Ethical Review Application Form</div></div>	
<p><i>Please use the Companion Guide to Completing the HREC Application Form and note that in order to complete this form correctly, you will need to read the HREC Guideline documents – specifically HREC Docs 4, 5 and 6 and the UCD Data Protection Policy and UCD IT Security Policies. Please see www.ucd.ie/researchethics for all guidelines and policies. . Please do not submit this form in PDF or docx format. Please put answers into the boxes provided and do not alter the format of this form.</i></p>	
Part A: Research Investigator(s)	
<p>1. Has this proposal been submitted to any other research ethics committee? (please tick appropriate box by double clicking and then checking)</p> <div style="display: flex; justify-content: space-around;"><input type="checkbox"/> Yes<input checked="" type="checkbox"/> No</div>	
<p>If yes, which committee and what was the outcome?</p> <div style="border: 1px solid black; padding: 2px; min-height: 1.2em;">n/a</div>	
<p>2. Is this a pilot study?</p> <div style="display: flex; justify-content: space-around;"><input type="checkbox"/> Yes<input checked="" type="checkbox"/> No</div>	
<p>3. Have you attended a one-to-one session with the Research Ethics Application Advisory Service?</p> <div style="display: flex; justify-content: space-around;"><input type="checkbox"/> Yes<input checked="" type="checkbox"/> No</div>	
<p>4. Short Title of Proposed Research for which approval is being sought</p> <div style="border: 1px solid black; padding: 2px; min-height: 1.2em;">Previous hamstring injury: associated lumbopelvic neuromotor activation levels and sagittal postural characteristics of running. A case control study</div>	
<p>5. Principal Investigator /Applicant</p> <p>a. Name (please include title if applicable)</p> <div style="border: 1px solid black; padding: 2px; min-height: 1.2em;">Colm Daly</div>	
<p>b. Position in UCD (please tick appropriate response)</p> <div style="display: flex; justify-content: space-around;"><input type="checkbox"/> Staff<input checked="" type="checkbox"/> Postgraduate<input type="checkbox"/> Undergraduate</div> <div style="border: 1px solid black; padding: 2px; min-height: 1.2em;">*Please note: the principal investigator is a postgraduate student studying a Masters in Sports and Exercise Medicine at Queen Mary University of London, United Kingdom.</div>	
<p>c. Details of Academic / Professional Qualifications</p>	
<p>1</p> <p>Further information from: www.researchethics.ie or email: research.ethics@ucd.ie or phone: 716 4689</p>	

BSc Physiotherapy, Certificate in Orthopaedic Manual Therapy

- d. UCD Address of School for correspondence (*home addresses are not acceptable*)

UCD School of Public Health, Physiotherapy and Population Science, Health Sciences Centre, University College Dublin, Belfield, Dublin 4.

- e. UCD Telephone:

01-716 6517

- UCD E-mail:

c/o ulrik.mccarthypersson@ucd.ie

6. Recent relevant publications of principal investigator, if applicable

N/A

7. Co-Investigator(s), (if applicable)

- a. Names (please include title if applicable)

Dr. Dylan Morrissey

- b. Position in UCD (please tick appropriate response)

☐ Staff

☐ Postgraduate

☐ Undergraduate

Dr Morrissey is a consultant physiotherapist and senior lecturer at Queen Mary University of London, Mile End, London E1 4DG, United Kingdom

- c. Details of Academic / Professional Qualifications

PhD, MSc, MMACP, MCSP

8. Supervisor(s), (if applicable) *supervisors must be members of UCD Staff*

- a. Supervisor Name(s) (please include title if applicable)

Dr Ulrik McCarthy Persson

- b. UCD School & email address

UCD School of Public Health, Physiotherapy and Population Science, Health Sciences Centre, University College Dublin, Belfield, Dublin 4
ulrik.mccarthypersson@ucd.ie

- c. Details of Academic / Professional Qualifications

BSc MSc PhD

9. Is this research being presented for an academic qualification? (please tick)

☒ Yes

☐ No

If yes, please specify. a letter of endorsement from the supervisor for this study must accompany this form

As part of completion for MSc Sports and Exercise Medicine, Queen Mary University of London, Mile End, London E1 4DG, United Kingdom

10. Research funding (if applicable)

a. Details of funding agency and programme

This research has been approved for a €500 research bursary by the Eastern Branch of the Irish Society of Chartered Physiotherapists.

b. If funded commercially, are there any restrictions on the freedom of the researcher to publish the results? (please tick)

☐ Yes ☒ No

If yes, please explain why.

11. Insurance/Indemnity arrangements –please read the Guidelines on Insurance/Indemnity (you will need to contact the UCD Safety Officer safety@ucd.ie to ascertain whether your study will be/is covered)

Insurance will be required –A request for insurance will be submitted on receipt of my research ethics reference number.

Please note: if insurance is required you will need to include details as a supporting document to this application form. You may wish to submit your request for insurance after you have submitted this form and received your research ethics reference number – if so please ensure you quote the reference to the Safety Office.

12. Have you read following?:

a. the current Guidelines and Policies for Ethical Approval of Research Involving Human Subjects issued by the Human Research Ethics Committee? Please see the research ethics website: http://www.ucd.ie/researchethics/hrec_policies_and_guidelines.html

(please tick) ☒ Yes ☐ No

b. Have you read the UCD Data Protection Policy www.ucd.ie/dataprotection/policy

(please tick) ☒ Yes ☐ No

- c. the Data Protection Guidelines on Research in the Health Sector
http://www.dataprotection.ie/documents/guidance/Health_research.pdf

(please tick) ☒ Yes ☐ No ☐ not applicable

Please note: It is not sufficient to read a paper copy which may be out of date. The latest versions of the guidelines and policies are posted on the UCD website. Your application may not be considered if you have not read the latest version.

Part B: Research Proposal

13. Full Title of the proposed research

Previous hamstring injury and associated lumbo-pelvic motor activation and postural characteristics of running. A case control study

14. Has this topic been studied before? (please tick appropriate response)

☐ Yes ☒ No

If yes, why is an additional study needed?

N/a

15. Provide a brief description of research (*not more than 200 words in each of the boxes below*). The description must be presented in everyday or lay language and detail:

a) the aims and objectives of the study

Aim:

To identify muscle activation patterns and lumbo-pelvic postural characteristics present in senior club level hurlers who have returned to full participation in sport following hamstring injury.

Objectives:

1. Recruit 15-20 hurlers who have sustained a hamstring injury in the previous 24 months.
2. Recruit an equal number of control subjects with no prior history of hamstring injury. These controls will be matched to the previously injured participants with regard to their team, age and playing position.
3. Carry out a baseline assessment and interview of all participants to ensure they meet the inclusion criteria.
4. Obtain baseline measurements for each participant – age, weight, height, playing position, playing exposure, injury history
5. Measure the muscle activity levels by recording surface electromyographic (s-EMG) signal over the previously injured biceps femoris (hamstring), both gluteus maximus, rectus femoris (quadriceps).

external oblique (abdominal) and lumbar erector spinae (low back) muscles during an unloaded step up, prone back extension, sit-up and Nordic lunge manoeuvre. The measurements will be used to normalise and make sense of the EMG signals during data analysis

6. Assess muscle activity of same muscles during three 10 second periods when running on a treadmill at 10km/h, 15km/h and subjective maximal pace.
7. During the same period of running, assess the 3D motion of the pelvis, hip, knee and lumbar spine posture using the CODAmotion system. This will be achieved by attaching small markers to the pelvis and legs of participants. The position of these markers will be recorded while the participants run.
8. Analyse the data collected to determine whether difference exists between subjects with and without previous hamstring injury during the late swing phase (just before the foot comes in contact with the ground) of high speed running.
9. Write up and report on findings

b) the scientific/theoretical background of study

Hamstring injuries, especially biceps femoris strains, are common in sports involving exposure to sprinting. Recurrence rates remain high. Recent research has begun to identify muscle and postural deficits which may expose the hamstring to increased stretch and strain during the final moments before foot strike in high speed running. Repetitive exposure to this stretch/strain cycle may account for an initial injury, but may also explain why the injured athlete may go on to experience repeated re-injury. Indeed, although based on small numbers and a non-blinded, a single study (Sherry & Best 2007) found a significant reduction in recurrence rates of hamstring re-injury when rehabilitation included addressing agility and lumbo-pelvic postural deficits, when compared to a 'traditional' strengthening and stretching regime.

To date no study has assessed lumbo-pelvic muscle activity or lumbo-pelvic kinematics in a group of athletes with prior hamstring injury. Such data may provide key clues to indicate why a previously injured hamstring would re-injure, and may also inform better rehabilitative strategies by beginning to provide a basis to further research.

c) the research design

- This will be a quantitative study as it will involve the analysis of numerical data (readings recorded from surface electromyograph and motion analysis)
- Subjects will be assessed on just one occasion, that is, a case control design will be employed with an imposed experimental condition

d) the methods of data collection

- Baseline data for each participant – age, injury and sport/activity related history will be collected via direct interview.
- Height will be measured using a standard measuring device, weight using weighing scales.
- All data will be entered directly into a computer database.
- The subject will be asked to perform a five minute warm-up at a self-selected pace on a treadmill.
- Muscle activity of the previously injured biceps femoris, both gluteus maximus, both rectus femoris, both lumbar erector spinae, both external obliques will be measured by surface Electromyography. This involves the placement of recording electrodes on subject's skin at sites in accordance with SENIAM guidelines. Prior to the placement of these electrodes it is necessary to prepare the subjects' skin. This involves gently shaving the area around the electrode placement site, and cleansing the skin with isopropyl alcohol.
- Electrodes will be applied to the participant behind a screen to ensure their privacy should anyone else be present in the room.
- Three trials of s-EMG data collection will be carried out as the subject perform a bodyweight step-up, Nordic lunge, prone back extension and sit-up. The data collected during these activities will be used to normalise the EMG data collected during running.
- The subject will have motion analysis markers attached to the skin at various points on the low back, pelvis, leg and foot. These points are according to previously published research and the manufacturers guidelines.
- All markers/sensors and associated wiring will be secured using an adhesive sports tape and spray.
- Data will be collect for three 10 second periods while the subject runs at

10km/h, 15km/h and their subjectively chosen maximum pace.

- The subject will perform a five minute cool down, at a self-selected pace, after which all sensors/markers will be removed.

e) the size and composition of sample

- 15-20 subjects with a history of prior hamstring injury, confirmed by interview and who have returned to full pain free participation in sport.
- The same number of control subjects (without prior hamstring injury), matched for team, playing position, age and exposure to sport.
- All participants will be aged 18 years or over.

f) how the size of the sample was determined

Sample size was determined based on number require to produce data from which statistical difference could be identified (Statistical Power >0.8, Significance 95%) from previous research using kinematic and EMG methods similar to those employed in this study.

g) whether there will be a pilot study run initially

The methods employed in this study will be tested for feasibility and the assessor will be tested for his reliability prior to data collection

h) the methods of analysis to be used

Descriptive analysis will be employed to describe the population characteristics of the two groups; age, weight, height.

EMG data will be expressed as a 'normalised' percentage of the peak values obtained during squat (rectus femoris and gluteus maximus), single leg stiff knee deadlift (biceps femoris), prone back extension (erector spinae) and sit-up (external oblique). This is to account for intra-subject variance in signal amplitude. Foot strike will be calculated on the previously injured leg, by analysing the change in acceleration of the foot motion analysis marker.

Normalised EMG data will be analysed for each running speed and each participant in a 120ms period before foot strike over ten strides. The root mean squared of the EMG amplitudes will be calculated within four consecutive 30ms

timeframes for each muscle. Finally the EMG data will be expressed as a ratio between the previously injured biceps femoris and each of the eight other muscles. Analysis will then be performed to determine whether a statistically significant difference exists between these ratios within the test group and the control group.

The motion analysis data will be analysed within the same 120ms window before foot strike, and root mean squared values determined for sagittal pelvic tilt, low lumbar curve, hip flexion/extension and knee flexion/extension angles. Analysis will then be performed to determine whether a statistically significant difference exists between these ratios within the test group and the control group.

(See section (i) below for specification regarding the analysis that will be used).

i) whether formal statistical procedures will be used

Formal statistical procedures will be used. Data will be analysed offline using a custom written MATLAB programme.

Means and standard deviations will be calculated for age, weight, height.

The data collected during the running trials are continuous in nature which enables the use of parametric and non- parametric statistics. The Kolmogorov-Smirnov goodness-of-fit test will be used to determine whether the data collected is normally distributed (Palant, 2001).

If the data is found to be normally distributed, a three way ANOVA test will be employed to compare groups using time frame and group as the main effect and muscle activity/joint angle as the dependent variable.

j) the expertise available to the researcher/s for analysis of the data

The formal statistical procedures outlined in the above section will be undertaken using a custom written Matlab programme. Support is available through the UCD

School of Public Health, Physiotherapy and Population Science and also at the Sports and Exercise Medicine Department, Queen Mary, University of London.

k) the proposed starting date of research/study

March 1st 2012

l) the proposed duration of research/study

4 months

m) the proposed duration of the data collection

1 months

16. Please identify any ethical dilemma which may arise in the course of the study.

1. Ensuring informed consent.

- To address this I will ensure that potential participant has read the information sheet, has had sufficient time to reflect on this and has had an opportunity to ask questions.

2. The participant is required to wear shorts to allow for testing.

- Participants will be informed of this prior to entry into the study through the Information Leaflet. The participant will be required to expose the buttock region in order to allow placement of the Gluteus Maximus electrode. The study will be carried out in accordance with the European Core Standards in Physiotherapy Practice (2008) – ensuring the dignity and rights of patients' is upheld at all times.

3. Possible inconvenience for the subject in participating in this study.

- Participants will be made aware through the Information Leaflet of the time commitment involved in partaking in this study (1 hour) and where testing will take place (UCD Human movement laboratory, Health Science building, Belfield). Furthermore they will be made aware that they are under no obligation to participate and are able to withdraw from the study at any time without prejudice.

Part C: Research Participants: Risk, Harm, Selection and Consent

17. Please indicate (tick) the level of risk for research participants:

☐ Extreme risk ☐ High risk ☐ Some risk ☒ Minimal risk

Please indicate the steps that will be taken to control this risk or to address any harm associated with participation (e.g. debriefing procedures, etc.).

Risk of injury to participants

- Individuals who have sustained a hamstring injury in the past are reported to be at increased risk of re-injury, especially when performing sprinting activity. Within this study a number of strategies are employed to reduce the risk of re-injury

- (a) All subjects are selected from a population who have returned to full participation in sport and have not recently sustained an injury.
- (b) All subjects will be screened for injuries/cardiac risk etc. prior to inclusion
- (c) All subjects will be required to undertake a warm-up procedure prior to testing
- (d) Maximum running pace will be subjectively selected by the subject
- (e) The exposure to maximum pace will be for a limited time period (approximately 15 seconds)

Falling on treadmill:

- Subjects will be allowed time to become accustomed to the treadmill prior to increasing the speed for testing. All wiring will be securely fastened with tape. An emergency stop cord will be attached to the subject's body.

Skin reaction:

Some individuals experience a mild transient skin irritation with the use of adhesive electrodes/markers. Every effort will be taken to avoid this, with short exposure times and use of hypoallergenic materials.

A fully qualified chartered physiotherapist will be present at all times during testing to provide appropriate management and advice in the unlikely event of an injury occurring.

Inconvenience (see Q16)

18. Provide details on the participants of the study:

a. Subjects. Who will participate in the study?

Male hurlers, aged 18+ and currently members of a Senior A level Championship hurling panel in Dublin. One group will be comprised of those who have fully recovered from a previous hamstring injury, while another will consist of matched controls

b. Selection and Recruitment: How will the research participants in this study be selected, approached and recruited?

- Volunteers will be hurlers currently members of a Senior A club team in the Dublin region.
- A direct approach will be made to the managers of hurling teams in the Senior A division of the Dublin Hurling Championship via email/telephone.
- Should the team manager agree, a study outline will be sent to them to pass on to potential participants (players).
- Potential participants will be asked to contact the principle investigator, if they are interested in taking part in the study.

i. Please state clearly who will approach potential participants?

Volunteers will approach the researcher through the contact details listed on the study outline, which will be given to them by their team manager.

c. Screening criteria for recruitment/selection of participants

i. Inclusion criteria. What inclusion criteria operate?

Inclusion and exclusion criteria for all subjects will be assessed via interview and brief physical assessment.

Hamstring Group:

- Subjective history of hamstring injury within 2 years, with specific features suggestive of definitive injury; pain location, sudden onset during rapid locomotion, pain on muscle contraction/running for 48hours+, inability to participate in sport for 48hours+.

- Participant has returned to full participation in sport without problems

All participants

- Currently a member of a Senior A Dublin hurling championship panel
- Negative results on the Physical Activity Readiness Questionnaire

ii. Exclusion criteria. What exclusion criteria operate?

- Pathology or previous surgery involving the spine, pelvis or lower limb
- Residual stiffness/weakness of the hamstring.

d. Vulnerable participants: *If the participants (or controls) belong to any of the following vulnerable groups please give details.*

i. Children under 18 years of age.

Not applicable

ii. University Students (see policies – accessing students and recommendations on using students in research)

University undergraduate/postgraduate students partaking in the Dublin Senior A Hurling Championship as member of a hurling team.

iii. People who have language difficulty

Not applicable

iv. People who have a recognised or diagnosed intellectual or mental impairment

Not applicable

v. Elderly people

Not applicable

vi. People confined to institutions (prisoners, residents in 24 hour nursing facilities)

Not applicable

vii. Persons in unequal relationships with the researcher (teacher/student, therapist/client, employer/employee)

Not applicable

viii. Others (please specify)

Members of a hurling team who, by virtue of peer/manager influence, may be feeling under pressure to participate.

19. If the study participants (or controls) belong to any of the vulnerable groups please state what special arrangements will be made to deal with issues of consent/assent.

The participants will be informed that involvement is voluntary and that no negative effects with regards to their study at UCD will occur.

The participants will be informed that there are no disadvantages or penalties for

partaking or withdrawing. This information will be conveyed through the information leaflet and also by the investigator.

20. Please confirm (right click to tick) that the following issues have been addressed in your Information leaflet for participants

	Tick
1. Introductory statement	
• Researcher's name and descriptor (Professor, Ms., Mr.)	<input checked="" type="checkbox"/>
• Name of researcher's School	
• The topic and title of the research.	
2. What is this research about?	<input checked="" type="checkbox"/>
3. Why are you doing this research?	<input checked="" type="checkbox"/>
4. How will the data be used?	<input checked="" type="checkbox"/>
5. What will happen if they decide to take part in this research study?	<input checked="" type="checkbox"/>
6. How will you protect their privacy?	<input checked="" type="checkbox"/>
7. What are the benefits of taking part in this research study?	<input checked="" type="checkbox"/>
8. What are the risks of taking part in this research study?	<input checked="" type="checkbox"/>
9. Can they change my mind at any stage and withdraw from the study?	<input checked="" type="checkbox"/>
10. How will they find out what happens with this project?	<input checked="" type="checkbox"/>
11. Contact details for further information	<input checked="" type="checkbox"/>

If not included in the information leaflet fully explain and justify why?

21. Describe the procedures by which consent will be obtained.

a. Is written consent to be obtained? (please tick)

<input checked="" type="checkbox"/> Yes	<input type="checkbox"/> No
---	-----------------------------

i. If yes, describe the procedures by which written consent will be obtained.

- Potential participant will be allowed seven days for reflection before signing the consent form.
- Participants' understanding of the extent of their role in the research will be accomplished by reading through the consent document with participants and discussing their participation before they become involved in the research.
- The potential participant will also be reminded that even after signing consent form they are still free to withdraw from the study at any time.
- The participant will also be encouraged to ask questions.

ii. If no, describe procedures regarding how consent will be obtained

--

Please send one electronic copy of the information sheet and the consent form for this submission together in one attachment.

22. Will payment of any kind, including expenses, be made to participants?

<input checked="" type="checkbox"/> Yes	<input type="checkbox"/> No
---	-----------------------------

If yes, please provide details and justification.

Participants will be asked to travel to the Human Performance Laboratory, Health Sciences Building, Belfield. They will be offered reimbursement for reasonable travel expenses from their home/workplace. Requests for reimbursement must be accompanied by a receipt of payment, a de-identified copy of which will be kept by the principle investigator.

Part D: Confidentiality and Data Protection

23. What arrangements are in place to ensure that the identity of each participant remains confidential?

Participants will be assigned a 5 digit coded number on entry to the study. The principal researcher (Colm Daly) and the research supervisor (Ulrik McCarthy Persson) will be the only individuals with access to the coding information.

24. Do you intend to use any of the following recording devices as a means of collecting information for this research study? (please tick)

- | | | |
|--|---|--|
| a. <u>Audio/Sound recorder (tape/cds)</u> | <input type="checkbox"/> Yes | <input checked="" type="checkbox"/> No |
| b. <u>Photography (incl. digital cameras/phones)</u> | <input type="checkbox"/> Yes | <input checked="" type="checkbox"/> No |
| c. <u>Film/Video/DVD recorder</u> | <input type="checkbox"/> Yes | <input checked="" type="checkbox"/> No |
| d. <u>Computer</u> | <input checked="" type="checkbox"/> Yes | <input type="checkbox"/> No |
| e. <u>Other</u> | <input type="checkbox"/> Yes | <input checked="" type="checkbox"/> No |

If **yes** is indicated for any of these devices, please indicate the specific permission that will be obtained as part of the informed consent document.

A detailed description of the data collection and storage procedures will be included on the information sheet. Participants will be encouraged to ask questions should they require any further clarification.

25. Please tick the form in which the data will be collected.

☐ Identified ☒ Potentially Identifiable ☐ De-Identified

26. Please tick the form in which the data will be stored and/or accessed.

☐ Identified ☒ Potentially Identifiable ☐ De-Identified

27. Describe the measures that will be taken to protect the confidentiality of the data which will be collected.

a. Who will have control of the data generated by the research?

Principal Investigator (Colm Daly) and Supervisor (Ulrik McCarthy Persson)

b. Please confirm where the data will be stored and that it complies with the guidelines.

Data will be securely stored on a password protected, 'hard drive encrypted'

external drive in accordance with the Data Protection Acts of 1988 and 2003.

c. In what format will the data be stored?

Data from testing and analysis will be stored on an Excel database on a password protected 'hard drive encrypted' external drive

d. For how long will the data be stored?

Data will be stored until October 2015 i.e. until completion of the study and 3 years after the study and publication has been written up

28. Responsibility for data collected in the study

a. Who will be responsible, until it has been destroyed, for the secure storage of and for control of access to the data generated by the research?

Supervisor Dr. Ulrik McCarthy Persson

b. Who will be responsible for destroying the data at the end of the period indicated in answer to Q 27d?

Supervisor Dr. Ulrik McCarthy Persson

c. Will the data be destroyed at or before the end of the study?

☐ Yes ☒ No

If yes, please confirm below that destruction has occurred in the Human Research Ethics Committee *End of Study Report Form* (HREC Doc 12).

If no, please indicate below how the person responsible for destroying the data will confirm to the Human Research Ethics Committee that this has occurred.

Supervisor Ulrik McCarthy Persson will notify the Human Research Ethics Committee via written correspondence.

29. Will any subsequent publication(s) entail the use of audio, video and/or photographic records? (please tick)

☐ Yes ☒ No

If yes, include how participants will be informed of this and confirm that this is included in the information leaflet and consent form.

Part E: Signed Declaration

30. Before the project can be given final approval by HREC (Sciences or Humanities), this section must be completed and submitted with original signatures.

Reference number:	
Title of research project:	
We the undersigned researchers acknowledge or agree with the University:	
<p>(a) It is our sole responsibility and obligation to comply with all domestic Irish and European legislation and to obtain such statutory consents as may be necessary.</p> <p>(b) Not to commence any research until any such consents have been obtained.</p> <p>(c) To furnish to the proper officer of UCD a true copy of any consent obtained.</p> <p>(d) That neither the University, the Committee, nor individual members of the Committee accept any legal obligation (to us or to any third party) in relation to the processing of this application or to any advice offered in respect of it nor for the subsequent supervision of the research.</p> <p>(e) That the research will be conducted in accordance with any approval granted by the Committee and in conformity with the documentation submitted with this application and with licence granted under any legislation.</p> <p>(f) That the undersigned researcher(s) have read the most recent UCD Research Ethics Committee Guidelines and Policy for Ethical Approval of Research Involving Humans – which are available on the UCD website (www.ucd.ie/researchethics) and agree to abide by them in conducting this research.</p> <p>(g) Confirm that the information provided on this form is correct and accurate.</p> <p>(h) In conducting research a researcher has both ethical duties and legal obligations. Compliance with one set of responsibilities does not guarantee compliance with the other - what is legally permissible may not be ethical and vice versa. It is for the researcher to inform himself and herself as to what ethical duties and legal obligations apply to his or her research and to comply with these duties and obligations.</p> <p>(i) It is not acceptable for an applicant to treat the grant of ethical approval as absolving them from the responsibility of informing themselves of their legal responsibilities in relation to data protection and of complying with these;</p> <p>(j) It must be understood that any ethical approval granted is premised on the assumption that the research will be carried out within the limits of the law;</p> <p>(k) Ethical approval does not constitute any sort of advice or representation to the applicant that compliance with the requirements, as laid down by the UCD Human Research Ethics Committee, will be sufficient to comply with the applicable law in the area.</p>	
Signature of Applicant:	Date:
Signature of Principal Investigator (if Applicant is not the P.I.):	Date:
Signature of Head of School (or designate):	Date:



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**Scoil na Sláinte Poiblí, na
Fisiteiripe agus Eolaíocht a
Daonra UCD**

Ionad Eolaíocht Sláinte,
Belfield, Baile Átha Cliath 4, Éire

physio.therapy@ucd.ie

Participant Information Leaflet

Previous Hamstring Injury: associated lumbopelvic neuromotor activation levels and sagittal postural characteristics of running. A case control study.

You are being invited to take part in a clinical research study. Before you decide whether or not you wish to take part, you should read the information provided below carefully and if you wish discuss it with your family, friends or GP. Take time to ask questions – do not feel rushed or under any obligation to make a hasty judgement. You should clearly understand the risks and benefits of participating in this study so that you can make a decision that is right for you – this process is known as Informed Consent.

You are not obliged to take part in this study. **You are free to participate.** You may change your mind, you are free to withdraw from this study at any time (before the start of the study or even after you have commenced the study) for whatever reason without having to justify your decision and without any negative impact.

WHY IS THIS STUDY BEING DONE?

Hamstring muscle strains/tears are common in sports which involve sprinting. Once you have torn your hamstring muscle you are much more likely to tear it again. We think that the muscle around your hips and pelvis help to protect the

hamstring during running. This may be due to the control they have on your posture. This study is examining if people who have had a hamstring injury in the past continue to have altered muscle activity and posture when we compare them to their teammates who never had this injury. This may provide clues for better rehabilitation programmes in the future.

WHO IS ORGANISING THIS STUDY?

The primary researcher is Mr Colm Daly, who is a chartered physiotherapist and postgraduate student. This study is being carried out in the School of Public Health, Physiotherapy and Population Science, University College Dublin and data collected will be analysed in Queen Mary University of London.

HOW WILL IT BE CARRIED OUT?

The study will be carried out from February 2012 to March 2012. Testing will take place in the Human Performance laboratory in the UCD Health Science Centre in Belfield campus, Dublin.

If you agree to participate, an appointment time suitable to you will be arranged.

WHAT WILL IT INVOLVE?

You should be aware this examination will take approximately **60 minutes** and will consist of the following:

- Firstly I will ask you some questions regarding previous injuries. Your height and weight will also be measured.
- As you will need to be appropriately dressed. For this you will need to remove clothing from your trunk (tee-shirt/shirt), and wear shorts, briefs, running shoes, ankle socks.
- The testing will involve the placement of self-adhesive electrodes on the skin over muscles on your back, buttocks, stomach and legs. We are looking at the activity of nine different muscles; biceps femoris (hamstring), both gluteus maximus (buttock), rectus femoris (quads), lumbar erector spinae (back) and external oblique (stomach) and we will mark the skin with a marker first. If the area is hairy, a small patch will need to be gently shaved.

- To mark the correct area over the gluteus maximus, your buttock area will need to temporarily be partly exposed. To ensure privacy, this will be done behind a screen.
- Before we attach anything you will complete a five minute warm-up on a treadmill.
- We will then attach the electrodes to the marked skin and also attach motion sensors to your pelvis, spine and leg and all the wires will be taped to keep them out of the way.
- In order to attach the electrodes to the skin over the gluteus maximus muscles, your buttock area will need to temporarily be partly exposed. To ensure privacy, this will be done behind a screen.
- You will then be asked to run on the treadmill for 10 seconds at each of the following speeds; 10km/hr, 15km/hr, and finally your maximum speed.
- You are free to withdraw from any of the tests included in the study while still remaining in the study, if so desired.

BENEFITS:

The main benefit from participating in the study is that the results yielded will help medical professionals to better understand the posture and muscle function of people who have had a previous hamstring injury.

RISKS:

There are some risks which you need to be made aware of before agreeing to take part.

- Running at full speed may increase the risks of you getting a muscle strain. As you have returned to full sporting activity, the risk is low, but never the less this risk remains.
- If you are not used to running on a treadmill, you may be in danger of tripping. Let us know if this is the case.
- Some people can get a temporary skin reaction to tape and shaving- this involves the skin becoming red and slightly irritated, and should clear up after one or two days.

WILL THERE BE ANY ADDITIONAL COSTS INVOLVED?

Reasonable travel expenses from your home/workplace in Dublin to University College Dublin will be reimbursed on production of a receipt. There are no other costs involved.

CONFIDENTIALITY ISSUES

All information which is collected about you during the course of the research will be kept **strictly confidential**. All information about you containing information as to your identity will be saved on encrypted files so that you cannot be recognised from it.

Each person that participates in the study will be given a code so it will not be possible to identify you once the information is collected. The only people with access to the coding information are the researcher (Colm Daly), and the research supervisor (Ulrik McCarthy Persson). Identifying information about you will not be used in any research reports. All information will be stored on a password protected and encrypted hard drive. When the study is completed, all information will be destroyed. The results of this study may be published in a scientific journal however none of the people who take part will be identified in any way.

HOW WILL I FIND OUT WHAT HAPPENS WITH THIS PROJECT?

If you would like to hear about the outcome of this research project please contact me using the telephone number or email address listed below.

IF YOU REQUIRE FURTHER INFORMATION NOW OR ANY FUTURE TIME PLEASE CONTACT:

Name: Colm Daly

Address: C/o Dr Ulrik McCarthy Persson, School of Public Health, Physiotherapy and Population Science, Health Sciences Centre, Belfield, Dublin 4

Email: colmedaly@hotmail.com Tel: 01 7166517

Name: Dr Ulrik McCarthy Persson

Address: School of Public Health, Physiotherapy and Population Science, Health Sciences Centre, Belfield, Dublin 4

Email: ulrik.mccarthypersson@ucd.ie Tel: 01 7166517



**UCD School of Public Health,
Physiotherapy and Population Science**

**ia Sláinte Poiblí, na
ripe agus Eolaíocht an
a UCD**

UCD Health Sciences Centre,
University College Dublin,
Belfield, Dublin 4, Ireland

Ionad Eolaíocht Sláinte,
Belfield, Baile Átha Cliath 4, Éire

T +353 1 716 6500

F +353 1 716 6501

physio.therapy@ucd.ie

Consent form

Please complete this form after you have read the Information Sheet and/or listened to an explanation about the research. If you have any question please contact me Colm Daly at **085 1017636** or **+44 7955391 677** or by email at **colmedaly@hotmail.com**

Title of Study: Previous hamstring injury: associated lumbopelvic motor activation levels and sagittal postural characteristics in running. A case control study.

Thank you for considering taking part in this research. The person organizing the research must explain the project to you before you agree to take part. If you have any questions arising from the Information Sheet or explanation already given to you, please ask the researcher before you decide whether to join in. You will be given a copy of this Consent Form to keep and refer to at any time.

I understand that if I decide at any other time during the research that I no longer wish to participate in this project, I can notify the researchers involved and be withdrawn from it immediately. No further data will be collected or used after this point in time. I consent to the processing of my personal information for the purposes of this research study. I understand that such information will be treated as strictly confidential and handled in accordance with the provisions of the Data Protection Acts 1988 and 2003.

Participant's Statement: I _____ agree that the research project named above has been explained to me to my satisfaction and I agree to take part in the study. I have read both the notes written above and the Information Sheet about the project, and understand what the research study involves.

Signed: Date:

Investigator's Statement: I _____ confirm that I have carefully explained the nature, demands and any foreseeable risks (where applicable) of the proposed research to the volunteer.

9.5.2 Observational study 2 (Nordics)

For Office Use Only:

Rec Reference

Date received:



Application form – Queen Mary Ethics of Research Committee

1 Name, department and email address of applicant

Colm Daly, PhD Researcher,
Centre for Sports and Exercise Medicine,
William Harvey Research Institute,
Bart's and the London School of Medicine and Dentistry,
Queen Mary University of London,
Mile End Hospital,
Bancroft Road,
London E1 4DG
c.daly@qmul.ac.uk

2 Title of study

Hamstring Associated Movement and Muscle activity deficits following Injury (HAMMI): The Nordic Project.

Running Title: Strengthening the hamstrings after muscle injury – an observational study.

3 Investigators

Colm Daly MSc COMT MCSP MISCP MHCPC - PhD Student, principal investigator,
Centre for Sports and Exercise Medicine, William Harvey Research Institute.
c.daly@qmul.ac.uk

<p>Dr Dylan Morrissey PhD MSc MMACP MCSP – Lead research supervisor, Centre for Sports and Exercise Medicine, William Harvey Research Institute. d.morrissey@qmul.ac.uk</p> <p>Athanasios Siouras– co-investigator, MSc (Sports and Exercise Medicine) student, Centre for Sports and Exercise Medicine, William Harvey Research Institute. a.siouras@smd13.qmul.ac.uk</p> <p>Robert Livingston, – co-investigator. iBSc (Sports and Exercise Medicine) student. Centre for Sports and Exercise Medicine, William Harvey Research Institute. RXL963@bham.ac.uk</p>
4 Proposed timetable
January 2014 – November 2015
5 Other organisations involved
Various sporting clubs/organisations according to written agreement when required.
6 Other REC approval
None
7 Nature of project e.g. undergraduate, postgraduate
Postgraduate PhD project with one MSc and one iBSc student project included
8 Purpose of the research
<p>Primary Aim</p> <ul style="list-style-type: none"> - The aim of this study is to explore the mechanisms of the Nordic exercise – a commonly used exercise in sports which has been shown to prevent hamstring injury and re-injury (Petersen et al 2011). <p>Secondary Aims</p> <ul style="list-style-type: none"> - To explore if those with prior HSI display statistically significant asymmetries in muscle activity during the Nordic exercise with comparison to their uninjured counterparts. - To determine whether such differences manifest as asymmetrical force production. - To establish whether the spatial pattern of muscle activation across the posterior thigh, measured using muscle mapping techniques, correlates with

injury site and time since injury.

- To determine if asymmetries in force production and spatial muscle activation are reversed following a 10 week Nordic training programme.
- To determine the long term effects of maintaining this programme in terms of injury prevention and biomechanical outcomes (1 year follow-up).
- To assess any differences between amateur and professional athletes.
- To develop a clinical measurement protocol, and associated database, to enable assessment of individual HSI athletes with respect to a normative database - for example generating a patient report and rehabilitation prescription.

Objectives:

1. Recruit 15-40 elite and sub-elite level athletes who have sustained a hamstring injury in the previous 24 months.
2. Recruit an equal number of control participants with no prior history of hamstring injury. These controls will be matched to the previously injured participants with regard to their team, sport, age and playing position where applicable.
3. Carryout a baseline assessment consisting of a brief physical assessment and interview of all participants to ensure they meet the inclusion/exclusion criteria. The physical assessment will consist of standardised measures of hamstring muscle function, neural dynamics, lumbar spine and sacro-iliac joint function. This assessment will be carried out by a qualified physiotherapist. Participants demonstrating exclusion criteria such as joint, muscle or neural dysfunction will be advised that they do not meet the criteria to take part in the study. An explanation of the assessment findings will be provided by the physiotherapist present and any beneficial advice provided.
4. Obtain baseline measurements for each participant – age, weight, height, playing position, playing exposure, injury history
5. Measure the athlete as they perform a Nordic Exercise. This will require the athlete to kneel on an instrumented platform with measurement hardware attached to their torso and legs.
6. The measurements will consist of the following
 - a. Measure the hamstring muscle activity by attaching an array of 32 electromyographic sensors to each leg.
 - b. Measure forces at the ankles by attaching load cells to ankle cuffs.
 - c. Measure ground force at the knees by placing a force plate on the platform
 - d. Measure 3D motion of the pelvis, hip, knee and lumbar spine posture using the CODAmotion system. This will be achieved by attaching small markers to the torso, pelvis and legs of participants. The position of these markers will be recorded while the participants perform the exercise.
7. Provide each athlete with a ten week training programme as described by Petersen et al (2011).
8. Complete repeat measures as described at 5 weeks, 10 weeks and 12 months.
9. Analyse the data collected to determine whether difference exists between

participants with and without previous hamstring injury during the Nordic exercise and where possible, correlate these findings with injury timing, site and severity.

10. Analyse the data collected to determine the effect of a ten week Nordic training programme (5 week, 10 week and 12 month follow-up) and determine if difference exists between participants with and without previous hamstring injury.
11. Write up and report on findings.

9 Study design, methodology and data analysis

Study Design:

- This will be a quantitative study as it will involve the analysis of numerical data (readings recorded from surface electromyograph, motion analysis and load cells).
- Participants will be assessed on four occasions, that is, a prospective cohort design will be employed with an imposed experimental condition.

Methodology

- Baseline data for each participant – age, injury and sport/activity related history will be collected via direct interview.
- Height will be measured using a standard measuring device, weight using weighing scales.
- All data will be entered directly into a computer database.
- Muscle activity of the hamstring muscles will be recorded by placing an array of 32 electromyographic sensing electrodes across both posterior thighs. Prior to the placement of these electrodes it is necessary to prepare the participants' skin. This involves gently shaving the area around the electrode placement site, and cleansing the skin with isopropyl alcohol.
- Electrodes will be applied to the participant behind a screen to ensure their privacy should anyone else be present in the room.
- The participant will have motion analysis markers attached to the skin at various points on the low back, pelvis, leg and foot. These points are according to previously published research and the manufacturers guidelines.
- All markers/sensors and associated wiring will be secured using an adhesive tape and spray.
- Participants will be asked to kneel on a cushioned platform incorporating a forceplate. Furthermore, instrumented ankle cuffs will be attached to their ankles (as previously described by Opar et al 2012).
- Force, electromyographic and movement data will be collected as the participant performs a Nordic exercise – the participant will lean forward from a kneeling position while keeping their back and hips straight. The ankle will be secured within the ankle cuffs. The participant will lean as far as possible and lower themselves to the ground using their hands to support them as they reach the floor. This will be repeated on three occasions
- We may also ask participants to perform a selection of additional exercises including a single leg deadlift with a 5kg weight.

- Participants will be asked to complete a ten week Nordic training programme (as described by Petersen et al 2011) under the guidance of their physiotherapist/coach if possible and with the support of a training diary.
- This 10 week programme consists of the following:
 - Week 1: A single session consisting of 2 sets of 5 Nordics.
 - Week 2: Two sessions consisting of 2 sets of 6 Nordics.
 - Week 3: Three sessions consisting of 3 sets of 6-8 Nordics.
 - Week 4: Three sessions consisting of 3 sets of 8-10 Nordics.
 - Weeks 5-10: Three sessions consisting of 3 sets of 12, 10 and 8 Nordics respectively.
- Repeat measures of muscle activity, force and movement (as described) will be completed at 5 weeks, 10 weeks and if possible at 1 year.

Data Analysis:

- Means and standard deviations will be calculated for age, weight, height
- EMG, force and motion analysis data will be collected in numeric form during each trial. This data will be used generate muscle activation maps of each hamstring and kinematic calculation relation to lower limb, torso and pelvic movement.
- Formal statistical procedures will be used and data will be analysed offline using a custom written MATLAB programme.
- The data collected during the running trials are continuous in nature which enables the use of parametric and non- parametric statistics.
- If the data is found to be normally distributed ANOVA tests will be employed to compare difference in groups by spatial location and timing of data collection.

10 Participants to be studied

Elite and amateur level team sport and track athletes, aged 18 years of age or more and current active members of a team and/or participating at a recognised competitive level. One group will be comprised of those who have fully recovered from a previous hamstring injury to the extent they have resumed sport, while another will consist of matched controls.

11 Selection criteria

Volunteers will be sought from elite and amateur level team sport and athletics organisations in the UK. Inclusion and exclusion criteria for all subjects will be assessed via interview and brief physical assessment.

Inclusion criteria:

Hamstring Group:

- Subjective history of hamstring injury within 3 years, with specific features suggestive of definitive injury; pain location, sudden onset during rapid locomotion, pain on muscle contraction/running for 48hours+, inability to

<p>participate in sport for 48hours+.</p> <ul style="list-style-type: none"> - Participant has returned to full participation in their sport without problems <p>All participants</p> <ul style="list-style-type: none"> - Currently a member of a team which compete at a nationally recognised level. - Negative results on the Physical Activity Readiness Questionnaire <p>Exclusion criteria:</p> <ul style="list-style-type: none"> - Current pathology or previous surgery involving the spine, pelvis or lower limb within two years of participation. - Residual stiffness/weakness of the hamstring.
<p>12 Recruitment (including incentives and compensation)</p>
<ul style="list-style-type: none"> • A direct approach will be made to the managers, coaching and/or medical support staff of elite and amateur level teams/organisations via email/telephone (see Appendix 2). • Should the managers, coaching and/or medical support staff agree, letters of agreement will be obtained, following which a study outline will be sent to pass on to potential participants (athletes). • Potential participants will be asked to contact the principle investigator or nominated researcher, if they are interested in taking part in the study. • Additional recruitment will be undertaken by displaying posters within private sports clinics, university campuses and sports clubs (see Appendix 1). Permission will be sought from management committees, owners etc. prior to displaying posters.
<p>13 Ethical considerations and risks to participants</p>
<ol style="list-style-type: none"> 1. Ensuring informed consent. To address this I will ensure that potential participant has read the information sheet, has had sufficient time to reflect on this and has had an opportunity to ask questions. 2. The participant is required to wear shorts to allow for testing. Participants will be informed of this prior to entry into the study through the Information Leaflet. The study will be carried out in accordance with the European Core Standards in Physiotherapy Practice (2008) – ensuring the dignity and rights of patients’ are upheld at all times. 3. Possible inconvenience for the subject in participating in this study. Participants will be made aware through the Information Leaflet of the time commitment involved for this study (90 minute testing session on four occasions and a 10 week training programme) and where testing will take place (QMUL

<p>Human Performance Laboratory, School of Engineering and Material Science, Bancroft Road). Furthermore they will be made aware that they are under no obligation to participate and are able to withdraw from the study at any time without prejudice.</p> <p>4. Risk of injury to participants</p> <p>a. Muscle soreness</p> <p>Some people suggest that the Nordic exercise programme may lead to temporary muscle soreness in the days after its completion – the Petersen programme is designed to allow participant time to become accustomed to the exercise ensuring early exposure is minimal with sufficient recovery time. In this way the risk of developing muscle soreness are minimal.</p> <p>b. Skin reaction:</p> <p>Some individuals experience a mild transient skin irritation with the use of adhesive electrodes/markers. Every effort will be taken to avoid this, with short exposure times and use of hypoallergenic materials.</p> <p>A fully qualified chartered physiotherapist will be present at all times during testing to provide appropriate management and advice in the unlikely event of an injury occurring</p>
<p>14 Confidentiality, anonymity, and data storage</p>
<ul style="list-style-type: none"> • Data from testing and analysis will be stored on an Excel database on a password protected 'hard drive encrypted' external drive. To ensure anonymity participants will be assigned a 5 digit coded number on entry to the study. The investigators (Colm Daly, Athanasios Siouras and Robert Livingston), and the research supervisor (Dylan Morrissey) will be the only individuals with access to the coding information. • A detailed description of the data collection and storage procedures will be included on the information sheet. Participants will be encouraged to ask questions should they require any further clarification
<p>15 Information for participants</p>
<ul style="list-style-type: none"> • Participants will be provided with an information sheet (see attached) which details the study purpose, methodology and data analysis procedures in a straight forward manner. • Subjects and will be given adequate time to read the information sheet. • Subjects will be invited to ask any questions and will be afforded the opportunity to make contact with a researcher after completion of the test protocol.
<p>16 Consent</p>
<ul style="list-style-type: none"> • Potential participants will be allowed adequate time for reflection before signing the consent form. • Participants' understanding of the extent of their role in the research will be

accomplished by reading through the consent document with participants and discussing their participation before they become involved in the research.

- The potential participant will also be reminded that even after signing consent form they are still free to withdraw from the study at any time.
- The participant will also be encouraged to ask questions.
- Consent will be obtained by a person, independent of club affiliations and in a neutral environment to ensure that the participant is free from any coercion.

17 Signature of applicant and authorising signatories.

Principal Investigator

Other Applicant(s)

(Head of Department)

Pro forma information sheet and consent form



Information sheet

Strengthening the hamstrings after muscle injury – an observational study.

Information for participants

We would like to invite you to be part of this research project, if you would like to. You should only agree to take part if you want to, it is entirely up to you. If you choose not to take part there won't be any disadvantages for you and you will hear no more about it.

Please read the following information carefully before you decide to take part; this will tell you why the research is being done and what you will be asked to do if you take part.

Please ask if there is anything that is not clear or if you would like more information.

If you decide to take part you will be asked to sign the attached form to say that you agree.

You are still free to withdraw at any time and without giving a reason.

WHY IS THIS STUDY BEING DONE?

Hamstring muscle strains/tears are common in sports which involve sprinting. Once you have torn your hamstring muscle you are much more likely to tear it again. The Nordic Exercise has been shown to help prevent hamstring injuries. This study is examining the effects of this exercise in previously injured and uninjured athletes. This information may allow us to develop better rehabilitation strategies to help prevent injuries in the future

WHO IS ORGANISING THIS STUDY?

The primary researcher is Mr Colm Daly, who is a chartered physiotherapist and postgraduate doctoral student.

HOW WILL IT BE CARRIED OUT?

The study will be carried out from January 2013 to November 2015. Testing will take place in the Human Performance Laboratory, Queen Mary University of London, Bancroft Road, London E1 4DG

If you agree to participate, an appointment time suitable to you will be arranged.

WHAT WILL IT INVOLVE?

You should be aware that we will ask you to come to the laboratory on four different occasions. The initial test will take place at a time of your convenience. The second test will take place five weeks later, the third will take place at 10 weeks and if possible a final test will take place after 1 year. Each test will take approximately *90 minutes*. You will also be asked to complete a training programme over the first 10 weeks.

The testing will consist of the following:

Firstly we will ask you some questions regarding previous injuries. Your height and weight will also be measured.

You will be asked some short questions regarding your general health. A physiotherapist will briefly check that you have full movement and strength in your hamstring muscle, as well as making sure you have no obviously problems with the flexibility of your nervous system, lower back and sacro-iliac joints. This is to ensure that it is safe for you to participate in this study, to make sure you can run without restriction/compensations and to in the case of participants who have had a previous hamstring injury, to help confirm that any pain you may have had previously was the result of a muscle injury.

If during the assessment the physiotherapist detects that you have any signs and/or symptoms which prevent you participating, no further data will be collected and an explanation of these findings will be provided. Should you wish, the physiotherapist will also be happy to provide you with any advice regarding these findings.

For testing, you will need to be appropriately dressed. For this you will need to remove clothing from your trunk (tee-shirt/shirt), and wear shorts, briefs, running shoes and ankle socks.

The testing will involve the placement of self-adhesive electrodes on the skin over hamstring muscles and we will mark the skin with a marker first. If the area is hairy, a small patch will need to be gently shaved. The skin will be lightly rubbed with a fine sand-paper and cleaned with an alcohol wipe.

We will then attach the electrodes to the marked skin and also attach motion sensors to your torso, pelvis, spine and leg and all the wires will be taped to keep them out of the way.

To ensure privacy, this will be done behind a screen.

We will ask you to knee on a cushioned platform and cuff will be attached to both your ankles.

You will then be asked to perform a selection of different exercise, one of which will be a Nordic – this will involve you leaning forward while keeping your back and hips straight. We may ask you to do this on three to four occasions to ensure you perform the exercise correctly.

We may also ask you to do a Single leg deadlift with a light weight.

We will provide you with a training diary which will outline a 10 week programme of Nordic exercises. We will be available to help you or your coach/physiotherapist at any stage throughout this period. The programme involves completing the exercise in the following way:

Week 1: A single session consisting of 2 sets of 5 Nordics.

Week 2: Two sessions consisting of 2 sets of 6 Nordics.

Week 3: Three sessions consisting of 3 sets of 6-8 Nordics.

Week 4: Three sessions consisting of 3 sets of 8-10 Nordics.

Weeks 5-10: Three sessions consisting of 3 sets of 12, 10 and 8 Nordics respectively.

You will be asked to come back for following up testing at 5 weeks and 10 weeks. If it is possible we would also like complete a final test after 1 year.

You are free to withdraw from any of the tests included in the study while still remaining in the study, if so desired.

BENEFITS:

The main benefit from participating in the study is that the results yielded will help medical professionals to better understand the muscle function of people who have had a previous hamstring injury when performing the Nordic exercise.

RISKS:

There are some risks which you need to be made aware of before agreeing to take part.

Some people suggest that the Nordic exercise programme may lead to temporary muscle soreness – the programme we use is specifically designed to allow you time to get accustomed to the exercise. In this way the risk of developing muscle soreness are minimal.

Some people can get a temporary skin reaction to tape and shaving- this involves the skin becoming red and slightly irritated, and should clear up after one or two days.

WILL THERE BE ANY ADDITIONAL COSTS INVOLVED?

There are no additional costs involved.

CONFIDENTIALITY ISSUES

All information which is collected about you during the course of the research will be kept strictly confidential. All information about you containing information as to your identity will be saved on encrypted files so that you cannot be recognised from it.

Each person that participates in the study will be given a code so it will not be possible to identify you once the information is collected. The only people with access to the coding information are the researchers. Identifying information about you will not be used in any research reports. All information will be stored on a password protected and encrypted hard drive. When the study is completed, all information linking your data and identity will be destroyed. We will keep the data for future analysis and comparison with other studied groups. The results of this study may be published in a scientific journal however none of the people who take part will be identified in any way.

HOW WILL I FIND OUT WHAT HAPPENS WITH THIS PROJECT?

If you would like to hear about the outcome of this research project please contact me using the telephone number or email address listed below.

IF YOU REQUIRE FURTHER INFORMATION NOW OR ANY FUTURE TIME PLEASE CONTACT US AT THE FOLLOWING:

For all general enquires relating to the research please contact:

Colm Daly, PhD Student, Centre for Sports and Exercise Medicine, The Mile End Hospital, Bancroft Road, London E1 4DG. Email: c.daly@qmul.ac.uk Tel: +44 7955391667

For information specifically relating to Queen Mary, University of London please contact:

Dr Dylan Morrissey, Centre for Sports and Exercise Medicine, The Mile End Hospital, Bancroft Road, London E1 4DG. Email: d.morrissey@qmul.ac.uk Tel: +44 20 8223 8839

It is up to you to decide whether or not to take part. If you do decide to take part you will be given this information sheet to keep and be asked to sign a consent form.

If you have any questions or concerns about the manner in which the study was conducted please, in the first instance, contact the researcher responsible for the study. If this is unsuccessful, or not appropriate, please contact the Secretary at the Queen Mary Research Ethics Committee, Room W117, Queen's Building, Mile End Campus, Mile End Road, London or research-ethics@qmul.ac.uk.

Consent form

Please complete this form after you have read the Information Sheet and/or listened to an explanation about the research.

Title of Study:

Strengthening the hamstrings after muscle injury – an observational study.

Queen Mary Ethics of Research Committee Ref: 2011/07/01

- Thank you for considering taking part in this research. The person organizing the research must explain the project to you before you agree to take part.
- If you have any questions arising from the Information Sheet or explanation already given to you, please ask the researcher before you decide whether to join in. You will be given a copy of this Consent Form to keep and refer to at any time.

I understand that if I decide at any other time during the research that I no longer wish to participate in this project, I can notify the researchers involved and be withdrawn from it immediately.

I consent to the processing of my personal information for the purposes of this research study. I understand that such information will be treated as strictly confidential and handled in accordance with the provisions of the Data Protection Act 1998.

Participant's Statement:

I _____ agree that the research project named above has been explained to me to my satisfaction and I agree to take part in the study. I have read both the notes written above and the Information Sheet about the project, and understand what the research study involves.

Signed:

Date:

Investigator's Statement:

I _____ confirm that I have carefully explained the nature, demands and any foreseeable risks (where applicable) of the proposed research to the volunteer

9.5.3 Case Reports

For Office Use Only:

Rec Reference

Date received:



Application form – Queen Mary Research Ethics Committee

1 Name, department and email address of applicant
Colm Daly MSc BSc COMT MCSP MISC PhD Student, Centre for Sports and Exercise Medicine, William Harvey Research Institute, Queen Mary University of London, Barts and the London School of Medicine and Dentistry, The Mile End Hospital, Bancroft Road, London E1 4DG. Email: c.daly@qmul.ac.uk Tel: +44 7955 391 667
2 Title of study
Hamstring muscle recovery following injury.
3 Investigators
Colm Daly MSc BSc COMT MCSP MISC – Lead investigator, applicant and PhD researcher, Centre for Sports and Exercise Medicine, William Harvey Research Institute. Dr Dylan Morrissey PhD MSc MMACP MCSP – Lead research supervisor, Centre for Sports and Exercise Medicine, William Harvey Research Institute.
4 Proposed timetable
Preferred Start Date: September 2014 Projected date of completion: Sept 2015
5 Other organisations involved
Various sporting clubs according to written agreement when required.
6 Other REC approval

None
7 Nature of project e.g. undergraduate, postgraduate
This project is part of an ongoing PhD
8 Purpose of the research
<p>Aim: To identify muscle activation patterns during the immediate and long term phases of recovery following hamstring injury.</p> <p>Objectives:</p> <ul style="list-style-type: none"> • Recruit min 15-40 max elite level athletes who have sustained a hamstring injury within the previous 3 months. • Carryout a baseline assessment consisting of a brief physical assessment and interview of all participants to ensure they meet the inclusion/exclusion criteria. The physical assessment will consist of standardised measures of hamstring muscle function, neural dynamics, lumbar spine and sacro-iliac joint function. This assessment will be carried out by a qualified physiotherapist. Participants demonstrating exclusion criteria such as joint or obvious neural dysfunction will be advised that they do not meet the criteria to take part in the study. An explanation of the assessment findings will be provided by the physiotherapist present and any beneficial advice provided. • Obtain baseline measurements for each participant – age, weight, height, playing position, playing exposure, injury history • Obtain injury/treatment history from the treating physiotherapist. • Obtain a copy of an imaging (MRI and/or ultrasound) if available. • Measure muscle activity levels across the hamstring region of all participants by recording multi-channel surface electromyographic (s-EMG) signal over both hamstring muscles during a maximal, pain free isometric muscle contraction at 0, 45 and 90 degrees of knee flexion. • Take simultaneous measures of force production using a hand held dynamometer held in place with a fixed belt. • Analyse the data collected to determine whether variation in myoelectric activity exists considering the fixed factors of subject group, injured/uninjured limb, time since injury, rehabilitation progress and location of injury on imaging. • Write up and report on findings
9 Study design, methodology and data analysis
<p>Study Design:</p> <ul style="list-style-type: none"> • This will be a quantitative study as it will involve the analysis of numerical data (readings recorded from surface electromyograph and motion analysis) • Subjects will be assessed on a weekly basis from recruitment until return to full return to sport.

Methodology:

- Baseline data for each participant – age, injury and sport/activity related history will be collected via direct interview.
- Imaging results and injury/rehabilitation history will be obtained from the treating clinician.
- Height will be measured using a standard measuring device, weight using weighing scales.
- All data will be entered directly into a computer database.
- Muscle activity of the hamstrings will be measured by surface electromyography. This involves the placement of recording electrodes on subject's skin. Prior to the placement of these electrodes it is necessary to prepare the subjects' skin. This involves gently shaving the area around the electrode placement site, and cleansing the skin with isopropyl alcohol.
- Electrodes will be applied to the participant behind a screen to ensure their privacy should anyone else be present in the room.
- Three trials of s-EMG data collection will be carried out as the subject performs painless isometric muscle contractions while lying on their stomach (i.e. bending knee against fixed resistance)
- We will measure force production using a hand held dynamometer held in place with a fixed strap.
- All sensors and associated wiring will be secured using an adhesive tape and spray.

Data Analysis:

- Means and standard deviations will be calculated for age, weight, height
- EMG and motion analysis data will be collected in numeric form during each trial. This data will be used generate a spatial representation of muscle activation across the hamstring.
- Formal statistical procedures will be used and data will be analysed offline using a custom written MATLAB programme.
- The data collected during the trials are continuous in nature which enables the use of parametric and non- parametric statistics.
- If the data is found to be normally distributed ANOVA tests will be employed to compare difference in group and limb considering injury location, time since injury, rehabilitation progress as covariates

10 Participants to be studied

Elite level association football, rugby, track and Gaelic games athletes, aged 18 years of age or more and current active members of a team and/or participating at an elite national level who have sustained a recent (<3months) hamstring injury.

11 Selection criteria

Volunteers will be sought from elite level association football, rugby, athletics and Gaelic games teams/organisations in the UK and Ireland. Inclusion and exclusion criteria for all subjects will be assessed via interview and brief physical assessment.

Inclusion criteria:

- History of hamstring injury within 3 months, with specific features suggestive of definitive injury; pain location, sudden onset during rapid locomotion, pain on muscle contraction/running for 48hours+, inability to participate in sport for 48hours+.
- Currently a member of an elite level association football, rugby and Gaelic games team or, in the case of track athletics, compete at an elite national level.

Exclusion criteria:

- Current pathology or previous surgery involving the spine, pelvis or lower limb.

12 Recruitment (including incentives and compensation)

- A direct approach will be made to the managers, coaching and/or medical support staff of elite level association football, rugby, track athletic and Gaelic games teams/organisations via email/telephone.
- Should the managers, coaching and/or medical support staff agree, a study outline will be sent to them to pass on to potential participants (athletes).
- Potential participants will be asked to contact the principle investigator or nominated researcher, if they are interested in taking part in the study.
- Additional recruitment will be undertaken by displaying posters within private sports clinics, university campuses and sports clubs. Permission will be sought from management committees, owners etc. prior to displaying posters.

13 Ethical considerations and risks to participants**Ensuring informed consent.**

To address this I will ensure that potential participant has read the information sheet, has had sufficient time to reflect on this and has had an opportunity to ask questions.

The participant is required to wear shorts to allow for testing.

Participants will be informed of this prior to entry into the study through the Information Leaflet. The study will be carried out in accordance with the European Core Standards in Physiotherapy Practice (2008) – ensuring the dignity and rights of patients' are upheld at all times.

Possible inconvenience for the subject in participating in this study.

Participants will be made aware through the Information Leaflet of the time commitment involved for this study (45-60 mins) on a weekly basis and where testing will take place (QMUL Human Performance Laboratory, School of Engineering and Material Science, Bancroft Road). Furthermore they will be made aware that they are under no obligation to participate and are able to withdraw from the study at any time without prejudice.

Risk of injury to participants***Re-injury:***

Individuals who have sustained a hamstring injury in the past are reported to be at increased risk of re-injury, especially when performing maximal muscle contraction. Within this study a number of strategies are employed to reduce the risk of re-injury

<ul style="list-style-type: none"> ▪ All subjects will be asked to perform a muscle contract at their subjective, painfree maximum. The lack of pain is likely to ensure that the forces generated remain at sub-injurious levels. ▪ The measurement taken uses a technique commonly employed by physiotherapists to track recovery following muscle injury. As such, the participant will be asked to perform an activity which is not unusual and forms part of standard post injury screening. <p>Skin reaction:</p> <p>Some individuals experience a mild transient skin irritation with the use of adhesive electrodes/markers. Every effort will be taken to avoid this, with short exposure times and use of hypoallergenic materials.</p> <p>A fully qualified chartered physiotherapist will be present at all times during testing to provide appropriate management and advice in the unlikely event of an injury occurring.</p>
<p>14 Confidentiality, anonymity, and data storage</p>
<ul style="list-style-type: none"> • Data from testing and analysis will be stored on an Excel database on a password protected 'hard drive encrypted' external drive. • To ensure anonymity participants will be assigned a 5 digit coded number on entry to the study. The investigators (Colm Daly), and the research supervisors (Dylan Morrissey, Ulrik McCarthy Persson,) will be the only individuals with access to the coding information. • A detailed description of the data collection and storage procedures will be included on the information sheet. Participants will be encouraged to ask questions should they require any further clarification.
<p>15 Information for participants</p>
<ul style="list-style-type: none"> • Participants will be provided with an information sheet (see attached) which details the study purpose, methodology and data analysis procedures in a straightforward manner. • Subjects will be given adequate time to read the information sheet. • Subjects will be invited to ask any questions and will be afforded the opportunity to make contact with a researcher after completion of the test protocol.
<p>16 Consent</p>
<ul style="list-style-type: none"> • Potential participants will be allowed adequate time for reflection before signing the consent form. • Participants' understanding of the extent of their role in the research will be accomplished by reading through the consent document with participants and discussing their participation before they become involved in the research. • The potential participant will also be reminded that even after signing the consent form they are still free to withdraw from the study at any time. • The participant will also be encouraged to ask questions. • Consent will be obtained by a person, independent of club affiliations and in a

neutral environment to ensure that the participant is free from any coercion.
17 Signature of applicant and authorising signatories.
<div style="text-align: center;"> <u>Principal Investigator</u> <u>Other Applicant(s)</u> <u>(Head of Department)</u> </div>

Pro forma information sheet and consent form



Information sheet

Hamstring muscle recovery following injury.

Information for participants

We would like to invite you to be part of this research project, if you would like to. You should only agree to take part if you want to, it is entirely up to you. If you choose not to take part there won't be any disadvantages for you and you will hear no more about it.

Please read the following information carefully before you decide to take part; this will tell you why the research is being done and what you will be asked to do if you take part. Please ask if there is anything that is not clear or if you would like more information.

If you decide to take part you will be asked to sign the attached form to say that you agree.

You are still free to withdraw at any time and without giving a reason.

WHY IS THIS STUDY BEING DONE?

Hamstring muscle strains/tears are common in sports which involve sprinting. Once you have torn your hamstring muscle you are much more likely to tear it again. We think that one reason for this is that following an injury, the hamstring muscle remains dysfunctional, even after you return to sport. This study is examining how muscle activity changes during rehabilitation in people who have had a hamstring injury in the recent past. This may provide clues for better rehabilitation programmes in the future.

WHO IS ORGANISING THIS STUDY?

The primary researcher is Mr Colm Daly, who is a chartered physiotherapist and postgraduate doctoral student.

HOW WILL IT BE CARRIED OUT?

The study will be carried out from September 2014 to September 2015. Testing will take place in the Human Performance Laboratory, Queen Mary University of London, Bancroft Road, London E1 4DG

If you agree to participate, an appointment time suitable to you will be arranged.

WHAT WILL IT INVOLVE?

You should be aware this examination will take approximately **45 - 60 minutes** on a **weekly** basis until you have return to play. The testing will consist of the following:

- Firstly we will ask you some questions regarding previous injuries. Your height and weight will also be measured.
- You will be asked some short questions regarding your general health. A physiotherapist will briefly check that you have full movement and strength and function in your hamstring muscle, as well as making sure you have no obvious problems with the flexibility of your nervous system, lower back and sacro-iliac joints. This is to ensure that it is safe for you to participate in this study, and to help confirm that any pain you have is not because of any other problem.
- If during the assessment the physiotherapist detects that you have any signs and/or symptoms which prevent you participating, no further data will be collected and an explanation of these findings will be provided. Should you wish, the physiotherapist will also be happy to provide you with any advice regarding these findings.
- We will also ask your physiotherapist for a copy of any scans related to your injury and to outline your rehabilitation to date.
- For testing, you will need to be appropriately dressed. For this you will need to wear shorts, and take off your shoes/socks.
- The testing will involve the placement of self-adhesive electrodes on the skin over muscles on the back of your thighs. We will mark the skin with a marker first. If the area is hairy, the area will need to be gently shaved. The skin will be lightly rubbed with a fine sand-paper and cleaned with an alcohol wipe.
- We will then attach the electrodes to the marked area.
- To ensure privacy, this can be done behind a screen.
- We will ask you to lay on your stomach on a bed and push your heel as hard as possible into a force measuring device without causing any pain. We will repeat this in three different positions, three times on each leg.

- You are free to withdraw from any of the tests included in the study while still remaining in the study, if so desired.

BENEFITS:

The main benefit from participating in the study is that the results yielded will help medical professionals to better understand the posture and muscle function of people who have had a previous hamstring injury.

RISKS:

There are some risks which you need to be made aware of before agreeing to take part.

- Forceful contractions of you hamstring muscle could cause an injury. We will ask you to make sure you have no pain when performing this activity – if the movement is painfree it is unlikely to cause a problem.
- Some people can get a temporary skin reaction to tape and shaving- this involves the skin becoming red and slightly irritated, and should clear up after one or two days.

WILL THERE BE ANY ADDITIONAL COSTS INVOLVED?

There are no costs involved.

CONFIDENTIALITY ISSUES

All information which is collected about you during the course of the research will be kept **strictly confidential**. All information about you containing information as to your identity will be saved on encrypted files so that you cannot be recognised from it.

Each person that participates in the study will be given a code so it will not be possible to identify you once the information is collected. The only people with access to the coding information are the researchers. Identifying information about you will not be used in any research reports. All information will be stored on a password protected and encrypted hard drive. When the study is completed, all information linking your data and identity will be destroyed. We will keep the data for future analysis and comparison with other studied groups. The results of this study may be published in a scientific journal however none of the people who take part will be identified in any way.

HOW WILL I FIND OUT WHAT HAPPENS WITH THIS PROJECT?

If you would like to hear about the outcome of this research project please contact me using the telephone number or email address listed below.

**IF YOU REQUIRE FURTHER INFORMATION NOW OR ANY FUTURE TIME PLEASE CONTACT
US AT THE FOLLOWING:**

For all general enquires relating to the research please contact:

Name: Colm Daly

Address: PhD Student, Centre for Sports and Exercise Medicine, The Mile End
Hospital, Bancroft Road, London E1 4DG.

Email: c.daly@qmul.ac.uk Tel: +44 7955391667

OR

Dr Dylan Morrissey

Address: Centre for Sports and Exercise Medicine, The Mile End Hospital, Bancroft
Road, London E1 4DG.

Email: d.morrissey@qmul.ac.uk Tel: +44 20 8223 8839

**It is up to you to decide whether or not to take part. If you do decide to take part you will
be given this information sheet to keep and be asked to sign a consent form.**

If you have any questions or concerns about the manner in which the study was conducted please,
in the first instance, contact the researcher responsible for the study. If this is unsuccessful, or not
appropriate, please contact the Secretary at the Queen Mary Research Ethics Committee, Room
W117, Queen's Building, Mile End Campus, Mile End Road, London or research-ethics@qmul.ac.uk.

Consent form

Please complete this form after you have read the Information Sheet and/or listened to an explanation about the research.

Title of Study: Hamstring muscle recovery following injury

Queen Mary Research Ethics Committee Ref: _____

Thank you for considering taking part in this research. The person organizing the research must explain the project to you before you agree to take part.

If you have any questions arising from the Information Sheet or explanation already given to you, please ask the researcher before you decide whether to join in. You will be given a copy of this Consent Form to keep and refer to at any time.

I understand that if I decide at any other time during the research that I no longer wish to participate in this project, I can notify the researchers involved and be withdrawn from it immediately. No further data will be collected or used after this point in time.

I consent to the processing of my personal information for the purposes of this research study. I understand that such information will be treated as strictly confidential and handled in accordance with the provisions of the Data Protection Act 1998.

Participant's Statement:

I _____ agree that the research project named above has been explained to me to my satisfaction and I agree to take part in the study. I have read both the notes written above and the Information Sheet about the project, and understand what the research study involves.

Signed:

Date:

Investigator's Statement:

I _____ confirm that I have carefully explained the nature, demands and any foreseeable risks (where applicable) of the proposed research to the volunteer

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